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EDITORIAL

CARDIOSPASM

Many theories have been put forward to explain the pathogenesis and aetiology of cardiospasm, but it still remains an unexplained disease. Other names have been given to the condition to emphasize different points of view on pathogenesis; hence such terms as achalasia, idiopathic dilatation, mega-oesophagus are to be found in the literature. Cardiospasm remains as the most widely used name for the clinical syndrome in which dilatation or obstruction occurs in the oesophagus. The term is not new; it was suggested by von Mikulicz in 1882. Arthur Hurst proposed the name achalasia—absence of relaxation—in 1913; he considered there was a disturbance of Auerbach's plexus in the condition and no true spasm. Holt has recently reviewed the problem in some detail and neatly presents the landmarks in the history of cardiospasm.[1]

The assumption that there is a disturbance of Auerbach's plexus in the wall of the oesophagus has caused many authors to compare the condition with congenital megacolon. There is a difference, however, in that in the latter condition the deficiency in the myenteric plexus is a local one, whereas in cardiospasm deficiencies occur throughout the length of the oesophagus—or perhaps not at all; or the changes observed may be secondary to inflammatory changes in the oesophagus. The term 'idiopathic dilatation of the oesophagus' may still be best as indicating our present unsatisfactory understanding of the condition.

The disease is believed to occur more frequently in females. The symptoms cause patients to seek advice in the 3rd or 4th decade, or sometimes at a much earlier age. The commencing symptoms are dysphagia and regurgitation, and pain which may simulate angina pectoris is in some cases an initial symptom. The site of the pain may vary, but substernal burning of persistent character is a usual complaint once severe inflammation develops. Exacerbations of spasm may apparently be produced by psychogenic factors, e.g. embarrassment during meals or from the patient's awareness of foulness of breath due to decomposing food in the oesophagus. While some patients remain well nourished, weight is usually lost from the regurgitation of food and varying degrees of hypoproteinaemia, hypovitaminosis and anaemia occur.

The narrowed distal part of the oesophagus is of varying length; it is just above the cardiac orifice and

VAN DIE REDAKSIE

MAAGMONDKRAMP

Baie teorieë is al geopper om die ontstaan en die oorsaak van maagmondkramp te verklaar maar sonder sukses. Om die verskillende standpunte i.v.m. hierdie siekte-ontstaan te beklemtoon is ander name aan die kondisie gegee. In die literatuur vind ons dus name soos achalasia, idiopatiese verwyding en mega-esofagus. Maagmondkramp is die benaming wat egter die meeste byval vind vir die beskrywing van die kliniese sindroom wanneer verwyding of verstopping in die esofagus voorkom. Mikulicz het dit in 1882 gebruik. In 1913 het Arthur Hurst die naam achalasia (gebrek aan ontspanning) voorgestel; hy was die mening toegedaan dat daar in hierdie kondisie 'n steuring in die Auerbach-pleksus voorkom en nie 'n ware kramp nie. Holt[1] het onlangs 'n volledige oorsig van die probleem gegee en die mylpale in die geskiedenis duidelik aangewys.

Die veronderstelling dat die Auerbach-pleksus in die esofaguswand versteur is, het daartoe gelei dat baie skrywers die toestand met aangebore megacolon vergelyk. Daar is egter 'n verskil. By aangebore megacolon is dit 'n plaaslike gebrek in die miënteriale vleg terwyl by maagmondkramp die gebreke langs die hele esofagus voorkom of miskien glad nie voorkom nie; of die veranderings wat waargeneem is kan ondergeskik wees aan veranderings wat aan inflammasie in die esofagus te wyte is. Die bewoording ‚idiopatiese verwyding van die esofagus' is miskien die beste beskrywing van ons huidige ontoereikende kennis van hierdie siekte.

Sover bekend kom die siekte meer dikwels onder vrouens voor. Die simptome dwing pasiënte om in hul dertiger en veertiger jare advies te soek, somtyds nog baie vroeër. Slukmoeilikhede en terugvloeiing is vroeë simptome en somtyds is 'n pyn, wat dié van angina pectoris naboots, die eerste simptoom. Die pyn mag op afwisselende plekke voorkom maar as ernstige inflammasie eers aanwesig is word daar gewoonlik oor 'n aanhoudend brandende gevoel onder die borsbeen gekla. Oënskynlik kan psigogeenfaktore die kramp vererger, soos bv. verleentheid tydens maaltye of omdat die pasiënt bewus is dat sy asem sleg ruik as gevolg van kos wat in die esofagus ontbind. Sommige pasiënte bly goed gevoed maar die meeste pasiënte verloor gewig as gevolg van die terugvloeiing van kos. Hipoproteïenemie, hipovitaminosis en anemie kom in groter of minder mate voor.

may be near the diaphragmatic hiatus. The constricted portion is not of more than normal thickness but the part of the oesophagus proximal to it is dilated, hypertrophied and lengthened. A fusiform, flask-shaped or sigmoid distortion of the oesophagus may result in cases of long standing, with inflammation of varying degree as a further complication.

Diagnosis is established by radiography, but valuable information may be obtained from oesophagoscopy if this can be performed. While the majority of patients with cardiospasm derive benefit from non-surgical methods of treatment, such as a non-irritant diet, antispasmodic agents, antacids and psychotherapy, these are not curative. Dilatation or surgical measures become necessary to relieve obstruction and possibly restore normal function at the oesophagogastric junction. The relief of obstruction has not proved a great problem, but for proper correction of the disorder many operations have been designed. Reflux oesophagitis has frequently occurred after operative procedures for relief of the obstruction and is a most disabling condition, as uncomfortable as the original cardiospasm. Holt[1] has introduced an operation which is designed not only to relieve the obstruction but also to avoid this subsequent reflux.

1. Holt, C. J. (1954): Amer. J. Med. Sci., **228**, 218.

Die lengte van die vernoude distaal deel van die esofagus wissel; dit is net bo die maagmond geleë moontlik in die nabyheid van die diafragmatiese spleet. Die vernoude deel is gewoonlik van normale dikte maar dié deel van die esofagus proksimaal daaraan is verrek, verleng en oorvergroot. Gevalle van lang duur mag 'n verwronge spoelvormige, flesvormige of S-vormige esofagus ontwikkel en inflammasie mag as 'n verdere komplikasie voorkom.

Diagnose word deur radiografie bepaal maar as dit moontlik is kan waardevolle inligting met behulp van 'n slukdermkyker verkry word. Nie-chirurgiese behandeling soos antikrampmiddels, nie-irriterende dieet, teensure en psigoterapie kan vir die meerderheid pasiënte verligting bring maar sulke behandeling genees nie. Chirurgiese optrede sal nodig wees om die verstopping te verwyder en moontlik normale werking by die slukdermmaagaansluiting te herstel. Dit is nie 'n moeilike probleem om die verstopping te verwyder nie maar baie operasies is al beplan om die ongesteldheid behoorlik te genees. Ná operasie vir die verstopping kom reflux oesophagitis dikwels voor en dié toestand is net so ernstig as die oorspronklike maagmondkramp. Holt[1] het 'n operasie beplan, om die verstopping te verwyder, wat hierdie reflux uitskakel.

1. Holt, C. J. (1954): Amer. J. Med. Sci., **228**, 218.

THE SPECIALIST QUESTION

Since the last meetings of the Medical and Dental Council and of the Federal Council of the Medical Association which took place in September and October last, no further decisions have been taken on the subject of specialist practice.

The Medical Council met in September a few days after the announcement of the results of the questionnaire which had been submitted to the medical profession of South Africa (published in the *Journal* of 18 September 1954: **28**, 815). This was also the first meeting of the Medical Council since the passing of the 1954 Act which amended the Medical, Dental and Pharmacy Act so as to legalize specialist registration. The Medical Council took no final decision on this subject, but in the meantime they decided (1) to continue the registration of specialists as in the past, and (2) to appoint a committee of the Council to investigate the subject and report to the Council. They also decided to invite the Association's Federal Council to express to this committee their views on (*a*) the interpretation of the referendum and (*b*) what steps should be taken.

The Federal Council met in October. No decision was come to on these points, but it was resolved to ask the scrutineers to analyse the voting papers again according to whether the voters were registered specialists or not, and according to whether they were in town or country practice.

This the scrutineers have done and we publish their report in this issue (page 263). The results will be before the Federal Council when it meets in Cape Town in a few days' time. Federal Council will then give further consideration to the Medical Council's request for its views. The date of the meeting was fixed so that it should precede the meeting of the Medical and Dental Council which is to take place, also in Cape Town, on 21 March and following days, when any decisions which may have been taken by Federal Council will be known to the Medical Council.

Comparing the way the general practitioners and the specialists respectively voted, it will be seen that they voted in conspicuously opposite senses in answering the question whether the voter was in favour of a register of specialists only, a register of consultants only, or a register of specialists plus a register of consultants. The results expressed as percentages were as follows:

	In favour of		
	Specialist Register only	*Consultant Register only*	*Two Registers*
Urban GPs	13	73	13
Country GPs	15	66	19
Specialists	64	14	22
Others*	26	48	26
All voters	26	55	18

* Full-time medical officers, interns and practitioners overseas.

Thus the general practitioners voted more emphatically against the continuance of a register of specialists than did the voters as a whole, who comprised not only the general practitioners, but also the specialists and 'others' (interns, full-time medical officers, etc.). The difference

between the voting of the urban GPs and the rural GPs on this point was only slight. (It will be observed that this analysis does not include the 292 voters who were in favour of a reversion to the system which existed before the introduction of the specialist register in 1938).

On the question of a statutory *versus* a voluntary register the specialists were overwhelmingly in favour of the statutory register, but the votes of the general practitioners, both urban and rural, were in favour of the statutory register by a narrower majority.

The voting against domiciliary visiting by specialists was also overwhelming. and the word can be fairly applied both to the GPs (20 : 1) and the specialists (7 : 1).

On all the questions at issue the difference between the voting of urban GPs and that of the rural GPs was not great enough to carry any significance.

THE CARE OF THE AGED

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No problems are isolated entities. Those centering around the aged are merely part of a great socio-economic problem, but the disconcerting selfishness of our small selves prevents us from appreciating this. Long life without health and security is more than a personal tragedy, it is a major social evil that may well threaten national economy.

It has recently been computed [1] that in the U.S.A. today there are 13 million people over 65 years of age. In England in 1891 45% of the population were under 20 years of age and 7% over 60 years; in 1947 27% of the population were under 20 years of age and 15% over 60. It has been calculated that in 1977 one-third of the population in Britain will be over 65 years of age. A comparable increase in South Africa over the past 50 years is shown by the rise of those over 60 from about 3·4% to about 9·4% of the population.

The frequency with which the increasing average age of the populations of various countries is emphasized in the literature indicates the urgent need of facing the problems surrounding old age. It is submitted that 90% of deaths are premature through infections, illness and neglect during youth or middle age, and that increasing age may be largely attributable to the innovation of modern drugs such as penicillin, aureomycin, etc. 'Bigger and better' antibiotics may further prolong lives and thus make the problem still greater. The Union Government and Provincial Administrations insist on compulsory retirement of all their employees at 60 years of age, although some may be at the peak of their productivity and some may desire to continue work. It is interesting to note that the Government seriously contemplates raising the retiring age of male public servants from 60 to 63. Dr. Wilfred Wright in a recent issue of *The Lantern* stated: 'There can be no denying that conservatism, inertia, and loose thinking all operate against the placement of unemployed older persons.'

Cause of Ageing. Two diagonally opposed theories still exist, viz. that ageing is due to (*a*) the result of wear and exhaustion, and (*b*) structural involution through disuse. One fact, however, is certain, viz. that abuse and disease accelerate ageing. Whatever the aetiology of atherosclerosis, it is reasonable to suggest that senility is largely the result of its effects on various structures of the human body.

In 1936, less than 1% of 3 million people studied in America were chronic invalids; the people studied were of both sexes and all ages, but it is not stated whether they were a random selection of the population.[2] While disease, in its chronic form is most prevalent between the ages of 35 and 40, actual invalidism and disablement reach their peak far beyond these years. Rheumatism (arthropathies) tops the list, together with heart disease, high blood pressure and arteriosclerosis. As a cause of disability, however, these conditions are surpassed by mental and nervous diseases. Statistics reveal that invalidism and disablement have increased in the past 15 years; and the human wastage is inestimable. Age is an important factor in chronic disabilities, and it is probably accurate to say that some form of chronic disease either patent or hidden, occurs in practically every aged patient.

In 1953 Sheldon, in a special survey of elderly folk in Wolverhampton, found that 66% suffered from some degree of disability, 22·5%were capable of only limited locomotion, 8·5% were confined to rooms, and 2·5% were bedridden. It is assumed that the analysis refers to those over 60 years of age.

Appropriate statistics in South Africa are unfortunately not available but an intimate association with a chronic-sick hospital in the Transvaal for several years has disclosed some interesting facts. During a 3-year survey there were 722 applicants for admission to the hospital. Of these 50% were regarded as sociological problems and therefore not admitted. Of the 370 applicants who were in the 'aged' group (i.e. 61 years and over) half qualified for admission. To quote further statistics at this stage might cloud the main issue of this article; suffice it to say that the facts and figures indicated the gravity of the problems concerning the aged. They certainly revealed tragically 'man's inhumanity to man' in the abundant examples of efforts to relegate invalid aged relatives to the mercy of strangers or to discard them as unwanted animate bodies that had outlived their utility. A suggestion is made that this may be due to the diminishing sense of family responsibility encouraged by Governmental pampering. Further, it appears to be insufficiently appreciated that elderly patients are often capable of rehabilitation. In this connection one was struck by the fact that in the symposium on Rehabilita-

tion at the Medical Congress held in Port Elizabeth last year, except for references by one or two speakers, little stress was laid on the importance of rehabilitating oldsters.

Let us, for purposes of discussion, group the aged in 4 main categories:

1. Those requiring care in acute general hospitals
2. Those requiring care in long-term hospitals
3. The homeless aged wholely or partially capable of looking after themselves
4. The homeless disabled aged incapable of looking after themselves but who can be satisfactorily looked after by an attendant, not necessarily of the skilled nursing type.

1. CARE OF THE AGED IN ACUTE GENERAL HOSPITALS

A common, though often fallacious, practice is to ascribe diseases in the aged to 'getting on in years'.

The characteristic differences between diseases of youth and of senescence may be summarized here:

	Youth	*Senescence*
Aetiology:	Exogenous	Endogenous
	Obvious	Occult
	Single	Multiple
	Specific	Superimposed
	Recent	Often over the past
Onset:	Sudden and florid	Insidious
Course:	Immunizing	Non-protective
	Acute, Self limited	Chronic and prolonged.

Whilst the main ailments of elderly patients are usually of a slowly progressive and degenerative type, there are very few diseases, including whooping cough and the so-called specific fevers of childhood, that may not occur during senescence. True, certain conditions occur more frequently in the aged, but it is doubtful whether any particular disease is caused by old age.

In our experience at Edenvale Hospital, the average period of hospitalization in acute wards was 11 days for those under 60 years of age, and 17 days for those over 60. In a previous publication, I referred to the frequency with which one finds young patients with subacute bacterial endocarditis, other heart ailments, and diseases of academic interest, and young cases in which the diagnosis has not been finalized, occupying acute beds for many months without application having been made for their transfer to a chronic sick hospital—a privilege seldom extended to the aged. Whilst accepting that the recovery period in the aged is usually longer than in younger patients, it is felt that a large percentage of the elderly patients for whom treatment is sought in long-term hospitals should be cared for in acute hospitals. A few examples may help to demonstrate this point:

Heart Disease. In the elderly this is not always the result of arteriosclerosis and is often as responsive to acute medication as in the younger age-group.

Bone and Joint Pains. These, ascribed to age, have often after investigation revealed themselves as the result of faulty posture, gout, myelomatosis, or secondary metastases.

Hemiplegia. As a disablement, this rates amongst the greatest wastages of man power. National figures are not available, but the importance of this disability may be gauged from the following:

In a recent survey W. J. Davison stated that in Western Australia 2.1 beds per thousand population were required for the care of hemiplegia and that 9% of all his chronic cases were hemiplegics. Our own figures showed that 173 of a total of 722 applications to a chronic sick hospital were for hemiplegics.

Recovery of movement in most hemiplegics is at best incomplete, and one feels that the only period during which treatment is of real rehabilitative value is shortly after the onset of the paralysis. On our experience, those admitted to non-acute hospitals never regained any useful or permanent activity. It is therefore submitted, in spite of overwhelming views to the contrary, that this group should receive treatment in an acute hospital. Incidentally we remain unimpressed with the results of anticoagulant therapy, so enthusiastically advocated by some.

Diabetes. While common at all ages—and it is known that barely 50% manifest symptoms of the disease—all the 57 applicants for admission to a long-term hospital in the Transvaal were over 65 years of age.

Gall-Bladder Disease. Common from 40 years upwards, not one of the 63 applicants, as chronics, was below the age of 68 years. It must be assumed that the younger sufferers of the 2 last-mentioned diseases had received medical attention in some acute hospital.

2. CARE OF THE AGED SICK IN LONG-TERM GENERAL HOSPITALS

Non-acute hospitals are specifically intended for those requiring prolonged and constant skilled nursing and/or medical attention, and *not* for incapacitated individuals who can be satisfactorily cared for by an unskilled attendant.

An analysis of 7,941 old chronic cases at the Peter Bent Brigham Hospital showed hypertensive disease (including 15% with cerebral arteriosclerosis) in 80%, neuropsychiatric disease in 59%, heart disease in 55%, gall-bladder disease in 25%, neoplastic disease in 14%, respiratory disease in 13%, diabetic disease in 10%, and malnutrition in 54%. Whilst practically every one over 50 years of age had osteo-arthritic disease, barely 10% complained of symptoms. Only a small percentage of these required prolonged hospitalization.

Data in a recent publication of mine [3] showed that the needs of the so-called chronics in the Transvaal are being satisfactorily served by the long-term section of Edenvale Hospital. Hitherto, however, inadequate facilities for rehabilitation have influenced the period of stay in this hospital and have precluded the admission of some.

A large section of the public and medical profession insist that it is the unqualified duty of the Provincial Administration to care for the chronic sick in long-term institutions. Provided cases are carefully assessed, I am partly in agreement. If, however, Professor Delore's definition of chronics [4] is accepted, it must remain an open question. He regards 'chronics as long-term sick,

with a potentially long span of life'. At the long-term section of Edenvale Hospital, out of 223 deaths, 180 died within 3 months of admission. One is therefore influenced to agree with Dr. Hugo's suggestion that the general hospitals should—after careful screening—make provision for most of their own chronics. As for others there would appear to be no serious obstacle to the introduction in this country of a scheme based on the Philadelphia plan for the home-care of chronically-ill old folk.[5] The plan, in essence, is one employing part-time medical practitioners, nurses and visiting domestics to serve the long-term sick in their own homes, or in provided homes where they are domiciled.

3. THE HOMELESS AGED

Whilst old-age homes fill a great need in a community, one feels that their provision is, in principle, no more commendable than the charitable provision of shelter for any other homeless being. It is noteworthy for serious criticism that the Transvaal boasts of several small private institutions supposed to be for the aged, but which exclude those who are unable to look after their own toilet requirements. Some even make a condition that inmates should be able to do their own cooking; practically all require that applicants should not be bedridden.

Basically, it should be the unquestioned responsibility of the State to look after the needy aged; but this should not be permitted to influence private individuals to shelve their duties towards their families. Efforts should be made to enlighten the public on their bounden duty to accept more personal responsibility and to depend less on Governments and charitable bodies. Churches, as one of the most powerful influences, should awaken this sense in their communities.

Old folk, like trees, do not like to be uprooted and are as a rule happier in their accustomed surroundings. It has often been remarked that the welfare of the aged depends more on home surroundings than on the disability from which they may suffer. It may be for this very reason that many are better off in institutions. Some oldsters, for example, have entirely grown away from their families and are happier in the company of other old people.

Management. The general principles of the management of the aged are common to all the 4 groups mentioned. They will however, for convenience, be embodied in this category.

The aged in general are more deeply and warmly grateful than younger folk for assistance, kindness, and patience. V. H. Matthews states, 'If I were to give a civil post to persons who were to have the care of old people, I should first find out whether they liked old people'. The supreme tragedy of old age is awareness of uselessness and unwantedness. In a book entitled *You Are Younger than you Think* Martin Gumpert states that he believes many old people die of boredom. Institutionalized oldsters should therefore be given fixed duties in the form of light work within prescribed limits according to their capabilities. They should be made to feel important and to believe that their duties are essential to the institution and the community. Recreation and diversional and occupational amenities should be provided in the form of arts and crafts, games, concerts and religious facilities. An inflexible rule with us is that elderly people are not permitted to be kept too long in bed or even too long at rest.

Diet. There is normally a decreased need for calories in oldsters, because of restricted activity and decrease in weight and a normal reduction in their interest in food. This may be contributed to by the loss of sense of smell and taste, missing teeth etc., and by psychological or sociological factors. Not uncommonly, however, elderly people have enormous appetites, if permitted to eat all they can. At a recent London Congress of the International Association of Gerontology it was reported that an octogenarian ate without any ill effects, in one meal, a large loaf of bread, 3 oz. of margarine, 3 oz. of cheese and 3½ oz. of sugar.

Food requirements are said to diminish after the age of 50 at the rate of 3% to 5% per decade. An ample diet for an oldster is provided by 1,500 to 2,000 calories per day, to include one g. of protein per kg. body-weight. Fats should be used sparingly. Obesity is merely the result of over-eating, irrespective of whether the appetite is stimulated by physiological or pathological factors. In humans, as in experimental rats, obesity is not conducive to 'ripe old age'. Detrimental at all ages, it is particularly undesirable in old age, not alone because of its tendency to promote atherosclerosis, but also as an aggravating factor in diseases such as bronchitis and asthma and in hernias.

Sub-clinical, or even frank, avitaminosis—especially deficiencies of vatimin C, thiamin, riboflavin, etc.—are not uncommon in the aged. Deficiency of vitamin A is to a large extent regarded as the cause for the harsh dry skin with hard keratotic plugs projecting from the hair follicles in the arms and thighs. Professor G. H. Percival of Edinburgh University reminds us that 'at a great age the skin may exhibit an almost juvenile smoothness and pliability of texture', so it would appear that the characteristic tissue-paper-like appearance of the skin of elderly people may not be entirely due to ageing of the tissues. Enlargement of the heart, with swelling of the feet, tender calves, and disorientated mental states are often improved by giving vitamin B, and the common manifestation of haemorrhages under the skin and liver deficiencies may be due to lack of vitamins C and K.

It is interesting to speculate that the reason for old people not responding readily to stress situations is a lack of vitamin C, as the integrity of the adrenal glands depends largely on this vitamin.

In summary, the diet should be balanced and light with regular meals and regular intake of fluid.

Contrary to popular belief, the average oldster does not suffer from constipation, but a weekly mild laxative may be preferred.

4. THE DISABLED AGED

From the criteria laid down in group 2, it is obvious that there are a large number of disabled aged who do not qualify for admission to any recognized institution in the Transvaal. It is specifically for the care of these that I desire to make my most earnest plea. They consist

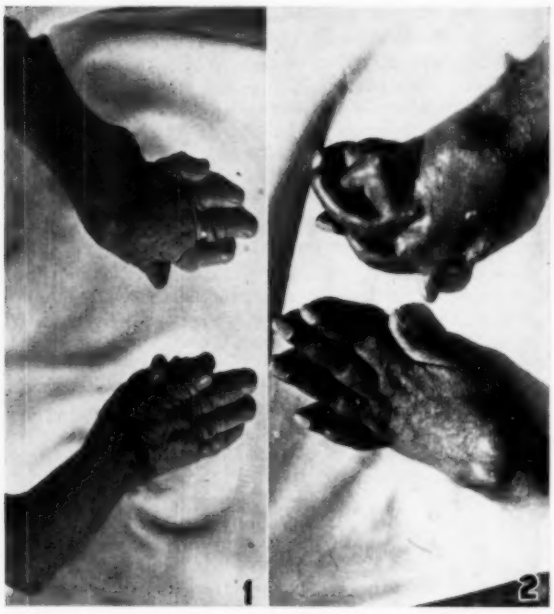
mainly of the crippled arthritics, sufferers from Parkinson's disease and limb deficiencies, hemiplegics and other paralytics, and senile incontinents. In spite of contrary views held by many, I submit that it should no more be the function of a technically or college trained individual to attend to the toilet requirements of an uncomplicated incontinent than that such specialized staff should be specifically engaged to wash, feed and dress non-sick cripples. I would add here, that a person should be regarded as bed-borne only if it is in the interests of his health that he should be confined to bed, and not because facilities are unavailable for getting him out of bed. It is an extraordinary thing that while legislation exists—even if only partially—to meet the needs of the sick, the aged, the healthy pensioner, and others, no provision is made to meet the needs of the disabled oldster who, had he but a home and the financial resources, could be satisfactorily looked after by a servant. Not only is there no legislative provision for this group, but even voluntary or charitable bodies appear to have forgotten them.

The accompanying photographs are but a few examples typifying incapacitated individuals who are generally regarded as chronic sick but could, it is submitted, be satisfactorily looked after by good domestic servants.

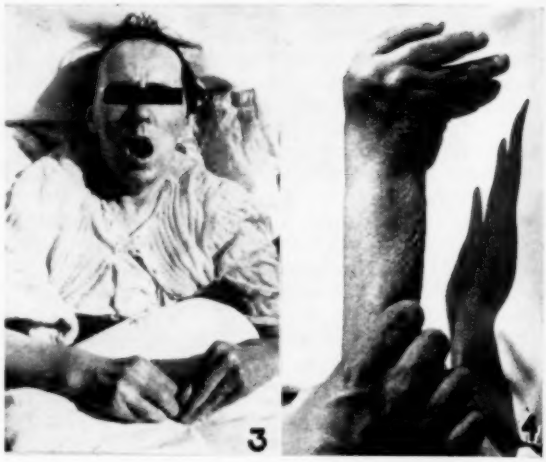
Fig. 1 shows the hands of a chair-borne old lady whose useless hands and feet—the result of a mutilating type of rheumatoid arthritis—prevent her from walking, feeding herself, or attending to her own toilet.

Fig. 2 shows the hands of a legless old gentleman suffering from a similar disease.

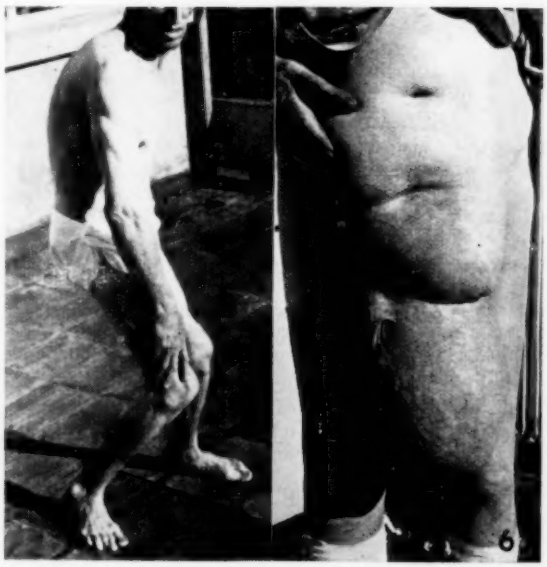
Fig. 3. This elderly lady, suffering from Parkinson's disease, is unable to walk unaided, dress or feed herself.



Figs. 1 *and* 2.



Figs. 3 *and* 4.



Figs. 5 *and* 6.

Fig. 4 shows the arm of a one-armed, leftsided hemiplegic old lady with rheumatoid arthritis, who is quite happy when placed in her chair.

Fig. 5. A youthful-looking 62-year-old congenital spastic, who had spent half a century in various institutions. He was not acceptable in a so-called old-age institution because of his facile mentally-arrested state.

Fig. 6. This 67-year-old subject with massive herniations and chronic heart disease had to leave a self-styled old-age home because he determinedly refused operation.

Dr. H. J. Hugo, Director of Hospital Services in the Transvaal, quoting extracts from the sixth Annual

Report of the National Corporation for the Care of Old People, says 'The Governors have for some years urged the provision of Homes for the Infirm or Rest Homes'. One wonders whether such homes are intended to provide for the disabled aged of this group or whether they are intended, once more, merely to act as old-age homes to assist children to 'park' their aged parents and relations.

CONCLUSION

To epitomize, the care of groups 1 and 2 (the aged sick in acute and long-term general hospitals) should remain the full responsibility of the Provincial Administrations, and that of groups 3 and 4 (the homeless aged and the disabled aged) shared between those members of the public on whom the responsibility falls, the State department of social welfare, and charitable bodies.

The care of group 4 (the disabled aged) should receive attention in priority to group 3 (the homeless aged not disabled). Both groups (3 and 4), however, might well be cared for in one institution, and I would suggest that consideration be given to the provision of such an institution, preferably with a trained qualified nurse constantly available. It would constitute a worthy effort for State-aided charitable bodies.

My thanks for the photographs are due to Mr. I. Norwich of the staff of Edenvale Hospital.

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REVISION SERIES

IX. SPRAINED ANKLE : THE DIAGNOSIS AND TREATMENT

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Most doctors, and many of the public, regard the sprained ankle as a minor ailment. It is thought to be an unfortunate and irritating accident and not to be classified as a major illness. The majority of sprains do in fact get well without treatment, but in some cases the pain and disability, like bad music, lingers on.

It is the object of this article to discuss the rational treatment of the sprained ankle, and its early diagnosis, and to show how correct treatment can accomplish a rapid and complete cure without residual disability or the resort to intensive, but rather ineffective, physiotherapy.

In discussing the many lesions that can appear either singly or together and form the clinical syndrome of the sprained ankle, it is necessary to specify exactly what is meant by a sprain. A sprain is strictly a minor lesion of a ligament. It consists of the tearing of a few fibres, the mass of the ligament being intact. If, on the other hand, the ligament is completely torn, then the condition is not a sprain but a rupture and the treatment of these two conditions differ widely.

The object in the treatment of a sprain is to eliminate pain and to accelerate healing while at the same time maintaining the mobility of the part. With a rupture no amount of immobilization is likely to secure healing and it will be necessary to consider surgical repair in order to reconstitute the completely torn ligament and to secure a sound result.

The differentiation between these conditions is relatively simple and there should be no confusion in the diagnosis. In the sprain, on stretching the sprained ligament, the increased tension gives rise to acute pain, since this structure is partially torn and its inflamed nerve endings are exquisitely sensitive; but in the rupture, on stretching the ruptured ligament, there is increased mobility but little pain, since the ligament is completely divided and there is therefore no tension.

In treating a sprain it is not necessary to completely immobilize the joint, since only a few fibres are torn and it is unlikely that further trauma will occur to tear the remainder of the fibres which go to make up the whole body of the ligament. It will be shown below how complete immobilization of the part leads only to avoidable trouble.

The different lesions which may be present in sprained ankle will now be considered:

THE LATERAL COLLATERAL LIGAMENT

The lateral collateral ligament is composed of 3 separate parts (Fig. 1):

(i) the anterior talo-fibular ligament running from the anterior aspect of the fibula to the neck of the talus,

(ii) the fibulo-calcaneal ligament running from the under surface of the fibula to the calcaneus, and

(iii) the posterior talo-fibular ligament running from the posterior aspect of the fibula backwards to the talus.

Sprain of the Distal Attachment of the Anterior Talo-fibular Ligament

Much the commonest sprain on the lateral side of the ankle-joint takes place at the distal attachment at the anterior talo-fibular ligament (Fig. 1 : 1). There is not much pain or disability associated with this lesion. The patient usually stumbles and twists his ankle and complains of pain over the neck of the talus. If the foot is plantar-flexed and inverted, so as to apply tension to the ligament (Fig. 2) the patient will complain of severe pain. Likewise pressure over the neck of the talus, at

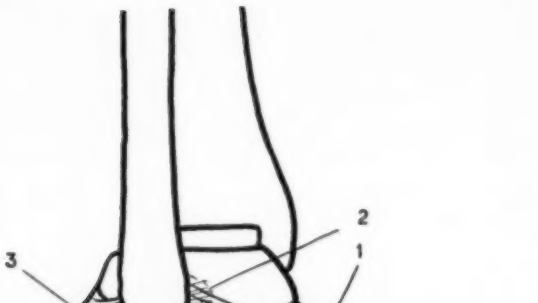

Fig. 1. The anatomy of the lateral collateral ligament of the right ankle-joint. 1. The site of the sprain at the distal end of the anterior talo-fibular ligament. 2. The site of the sprain at the proximal end of the anterior talo-fibular ligament. 3. The site of the sprain at the proximal attachment of the fibulo-calcaneal ligament.

the distal attachment of the ligament, will give rise to pain.

When the patient is seen first there may be considerable swelling around the ankle-joint and it is sometimes hard to differentiate a sprain from a fracture of the lateral maleolus. This can easily be accomplished by pressing the fibula at a higher level well above the area of swelling. If there is a fracture then crepitus will occur between the bony fragments, giving rise to acute pain at the fracture site. If, however, the patient is only suffering from a sprain, no unpleasant sensation will be felt.

Treatment. Immobilization is not required. The ankle may be strapped in eversion with extension plaster and elastoplast. The strapping will give a feeling of support and will also limit the swelling. The strapping should cover the foot from the base of the toes and it should extend two-thirds of the way up the calf. The patient should be instructed to carry on with normal exercise and should be encouraged to walk since this will definitely diminish the amount of the haematoma and oedema associated with the sprain. If the patient is active and carries out these instructions a cure can be effected within 5 days. Injections of local anaesthetic into the tender area can only offer temporary relief and will do nothing to materially hasten the process of healing.

Sprain of the Proximal Attachment of the Anterior Talo-fibular Ligament

The second most common area to be sprained is the proximal attachment of the anterior talo-fibular ligament (Fig. 1 : 2). Here a few fibres are torn from the lower end of the fibula giving rise to considerable pain. The patient is, however, able to walk after this injury until about 2 hours have elapsed, when increasing pain and stiffness make walking difficult.

The proximal attachment of this ligament is closely associated with the capsule of the joint and when it is sprained some trauma is transmitted to the capsule causing irritation and giving rise to an effusion. The patient will then complain of being unable to walk properly and will prefer to walk on his toes because the plantar-flexing of the ankle enlarges the cavity of the joint so that the fluid can be accommodated; if, however, the ankle is dorsi-flexed and weight is taken square on the foot, the capsule will be ballooned anteriorly, which gives rise to acute pain.

Aspiration of the ankle-joint at this stage can be performed and this gives immediate relief; but this measure is not often necessary. Strapping of the ankle joint in slight eversion will relieve most of the symptoms, and the patient should then be instructed to walk on the foot as much as possible.

Many practitioners regard a severe sprain as an indication for plaster-of-paris fixation which, initially, serves to give relief from pain; but the later results of treatment by this method are not free from complications. As the sprain heals and the haematoma organizes, adhesions will be formed between the ligament and the capsule of the joint. If, on the other hand, the patient continues to exercise the joint, one can expect a sound and satisfactory result in 3 weeks. The movement of the capsule adjacent to the sprain delays healing somewhat but there will be no adhesions. If, however, the patient has been treated in plaster or has not exercised the ankle during the period of healing, adhesions will form between the ligament and the capsule; then as the patient walks the capsule of the ankle-joint is irritated, which causes swelling of the joint, and he will complain of pain on exercise and swelling towards the end of the day.

Unfortunately, this type of lesion is commonly ignored by the practitioner, who considers a little massage sufficient to mobilize the joint. It is classified as 'an old sprain—not much one can do for it, it will get better on its own'. From this misdiagnosis, and the lack of improvement that follows, the patient eventually consults a chiropractor. The chiropractors have only one form of treatment and in such cases as these it is a most successful measure. They manipulate the joint, and when the adhesions are snapped the patient feels the ankle giving way and hears the click. He is then informed that a little bone has gone back into place, and as he is cured he is highly delighted and proceeds to broadcast the results of this 'miracle' to his friends.

The medical practitioner can never compete with the chiropractor until he appreciates the true nature of this lesion. He should realize that by his early treatment and advice he should keep the ankle-joint moving. If, however, he fails in this he should be the first to recognize the lesion and recommend manipulation of the joint, because by ignoring the condition he leaves the patient with considerable disability which is definitely remediable.

The Diagnosis of Adhesions. To make a correct diagnosis of adhesions before recommending manipulation, the practitioner should note:

(*a*) The history of trauma,

(*b*) the persistence of swelling and/or pain for more than 4 weeks,

(*c*) limitation of movement when stretching the affected ligament, and

(*d*) that tension on the affected ligament still gives rise to pain.

(*e*) An X-ray should be taken in order to exclude a fracture that may have been missed initially.

Manipulation of the Ankle-joint. This is preferably carried out under general anaesthesia. The ankle-joint should be moved once only through the full range of movement. The snapping of the adhesions will be felt quite distinctly. Following the manipulation the patient must be encouraged to move the joint to the limit of its normal range at least 3 times a day for a period of 3 weeks; otherwise the ligament will again become adherent to the capsule. Massage is valuable in the apprehensive patient because the masseur makes sure that the normal range is maintained.

Sprain of the Fibulo-calcaneal Ligament

The third and last lesion which occurs in the lateral collateral ligament is a sprain of the proximal attachment of the fibulo-calcaneal ligament (Fig. 1 : 3). This may be recognized by tenderness under the tip of the fibula and the complaint of pain on inversion of the foot with the ankle in the neutral position. This again takes 3 weeks to heal; it should be treated with strapping. Usually it gives rise to no disability although occasionally, if it has been treated in plaster, repeated swelling and pain can occur. The same remarks apply to this strain as to that of the anterior talo-fibular ligament and the patient should be instructed to keep the joint moving at all costs.

OTHER LIGAMENTS

Sprain of the Medial Collateral Ligament

The deltoid ligament, which is attached proximally to the under surface of the medial maleolus fans out and is attached distally to the talus and to the calcaneous. Sprains occur only at its proximal attachment, and these are rare. If, however, a sprain of this ligament is diagnosed by pain and tenderness under the tip of the medial maleolus (Fig. 3), and if this appears to be the sole lesion, a fracture of the fibula should be suspected; for it is nearly impossible, mechanically, to strain this ligament without some damage taking place on the outer side of the joint, because in the mortice of the ankle joint the fibula extends further distally than the medial maleolus.

Treatment. There is no need to consider the treatment of this lesion in a special light. Strapping of the joint in inversion is sufficient to relieve the patient of the acute symptoms. Occasionally there is an effusion present but usually the lesion is mild and active exercises should be instituted at once in order to avoid the formation of adhesions.

Sprain of the Tunnel of Tibialis Posticus

This may occur as a complicating factor in sprains of the ankle. The tenderness is, however, $\frac{1}{2}$ inch below the tip of the medial maleolus and should not be confused with that of sprains of the deltoid ligament. Abduction of the forefoot gives rise to pain, and tenderness over the tendon tunnel can easily be elicited. Occasionally one can also feel crepitus as the tendon moves in its tunnel.

The treatment here may be different. The patient is really suffering from teno-synovitis and in the acute stage an injection of hydro-cortone into the tendon sheath will often dramatically relieve the symptoms. If, however, the condition persists a tilt on the inner side of the heel of the shoe, to diminish the strain of the tendon on its tunnel, gives a very beneficial effect. Usually the condition heals within a few days.

Sprain of the Inferior Tibio-fibular Ligament

The mortice of the ankle joint is controlled by the inferior tibio-fibular ligament. This ligament, running

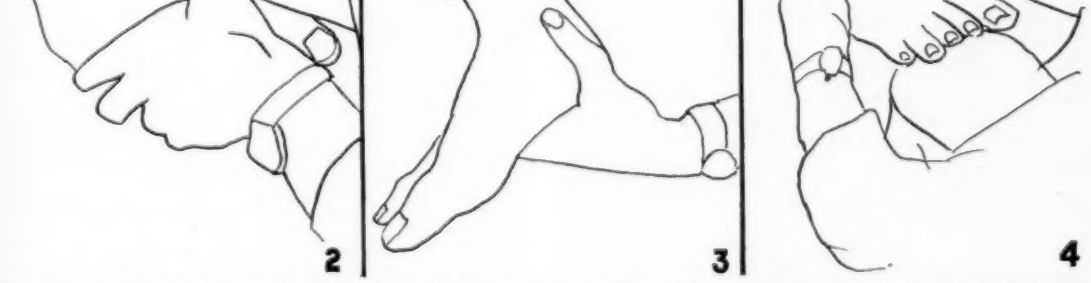

Fig. 2. The foot is forced into plantar flexion and inversion to test the anterior talo-fibular ligament. *Fig.* 3. Tenderness under the medial maleolus indicates a sprain of the medial collateral ligament. *Fig.* 4. Testing for a sprain of the inferior tibio-fibular ligament. The hands are compressing the maleoli and the knee is pushing the foot up into dorsi-flexion.

from the outer aspect of the lower end of the tibia to the fibula, holds the fibula in apposition and so keeps the ankle-joint together. Occasionally the patient, stumbling with the foot forced into hyper-dorsi-flexion will cause a strain of this ligament to take place.

In the young subject with a normal ankle, movement of the foot from plantar-flexion to dorsi-flexion separates the tibia from the fibula by a distance of $\frac{1}{8}$-$\frac{1}{4}$ inch and this is accomplished by the superior articulating surface of the talus, which is broader in front than behind. If, however, this wedge is forced still further between the bones, the sprain, or even rupture, of the interior tibio-fibular ligament will take place.

The patient complains of pain and tenderness in a somewhat similar area to that of a sprain of the proximal attachment of the anterior talo-fibular ligament, but the differential diagnosis can easily be made on examination. The examiner grips the patient's ankle in both hands and compresses the tibula and fibula. With his knee he then moves the foot up into dorsi-flexion just short of the position where the pain occurs (Fig. 4). He then suddenly releases his hands, allowing the tibia and the fibula to spring apart. If there is a sprain of the interior tibio-fibular ligament the patient will complain of acute pain.

Treatment. Again it is unnecessary to consider plaster-of-paris as a form of treatment since this will only give rise to adhesions because of the complete immobilization. The strain, however, must be taken off the ligament in order to secure healing. It is, therefore, necessary to raise the heel of the shoe about $\frac{1}{2}$-$\frac{3}{4}$ inch so that the narrower posterior portion of the superior surface of the talus is presented in the mortice. This is effective in securing union but tenderness may continue for 6 weeks.

Teno-synovitis of the Tendo Achillis

This may be a complicating factor in sprains of the ankle joint, particularly where strain has been placed on the Achilles tendon by forced dorsi-flexion. The patient complains of pain and tenderness over the tendo achillis and on palpation crepitus may often be felt. To relieve this, tension should be taken off the heel string and this can be done by raising the heel $\frac{1}{2}$-$\frac{3}{4}$ inch. Usually this is sufficient and in about 3 weeks the condition settles down. If, however, the patient complains of acute pain, injections of hydrocortone into the sheath of the tendo achillis will be effective in causing resolution.

Teno-synovitis of the Long Extensors of the Toes

Occasionally in sprained ankles, where the injury is severe, and the foot has been forced into hyper-plantar flexion a teno-synovitis may occur in the long extensors of the toes. This is characterized by two swellings, one above and one below the ankle joint. The sheaths of the extensors communicate under the annular ligament. Pressure on one swelling will give rise to an increase in size of the other, and this can be considered a diagnostic sign. No treatment is necessary and the condition rapidly clears up.

It should be noted that all these lesions may occur in different combinations. A severely sprained ankle is often a multiple lesion, and each component should be carefully diagnosed in order to give an opinion as to the prognosis. At no time in the treatment of the sprained ankle is plaster-of-paris necessary since this only encourages stiffening of the joint and the formation of adhesions in the area, which may cause the patient residual symptoms long after the original lesions have healed.

The sprained ankle may be a complex matter and should be considered in that light. It should not be regarded as a lesion of minor importance; the patient has the right to expect at least the same degree of competence and interest from his doctor as he would receive from an unqualified practitioner. If the profession regards this condition as unworthy of the exercise of normal clinical acumen and care, then it has little right to complain of competition from the 'quacks'.

INTERNAL HERNIA

REPORT OF TWO CASES

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The unique experience of discovering 2 cases of internal hernia, and the rarity of the condition, prompt me to report the cases. In my view they bring out certain differences, and I propose to submit evidence from the literature showing that there are 2 types of internal hernia, namely (1) into the paraduodenal fossa (right or left) and (2) retroperitoneal.

CASE REPORTS

Case 1. Mrs. E.v.S., aged 33 years, complained of a persistent pain in her right side since a Caesarean section 16 months previously. Her bowel acted regularly. She did not suffer from abdominal distension. On examination of the patient no abnormality other than tenderness in the right iliac fossa was found.

A barium-meal report by Dr. K. V. O. Gunn read, ' A few coils of jejunum appear to be in an internal sac; no obstruction exists'. The plate (Fig. 1) [1] brought out very clearly the translucent 'paper bag' containing small bowel, as described by Exner (see Lahey and Trevor [2]). A diagnosis of internal hernia was made. At operation I found all the small bowel except the terminal 12 inches of ileum within a peritoneal sac (Fig. 2). The opening of the sac, in the region of the ligament of Treitz, faced towards the left and easily admitted the closed fist. The small bowel was very easily withdrawn. I was able to close the neck of the sac without injury to the superior mesenteric vessels which lay in its free margin. The sac I obliterated by 'purse string' sutures in its anterior wall. Subsequent con-

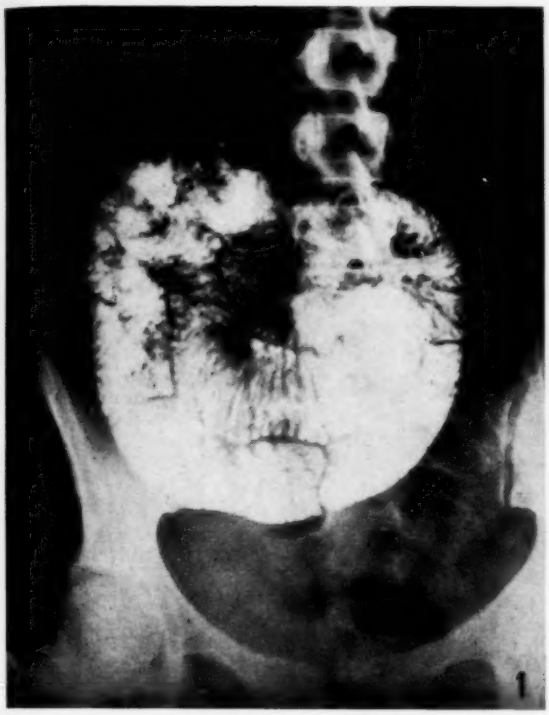

Fig. 1. Barium-meal X-ray of a right paraduodenal-fossa hernia showing characteristic translucent 'paper bag' containing small bowel. This picture was published on a larger scale in the *Journal* (1954): **28,** 516.[1]

valescence was normal, and a later X-ray revealed small bowel again free in the pelvis.

This case is one of hernia in the right paraduodenal fossa, corresponding to those described by Francis R. Brown [3] and by Lahey and Trevor.[2] Brown's case had a neck to the sac which admitted 4 fingers. He was able to turn the sac inside out, suture the neck, and remove the sac. Lahey and Trevor, at an exploratory operation in one of their 2 cases, revealed a herniation of the proximal jejunum through the right paraduodenal fossa. The jejunum was pulled out of the sac, its opening sutured, and a small portion of the jejunum then buttressed against the opening.

Case 2. A soldier V.A.T., aged 46 years, was admitted with acute abdominal pain. He stated he first experienced this generalized abdominal pain in the early hours of the previous day. Before this he had enjoyed good health except for constipation in the past 2 years. Later during the day of admission the pain became even more severe, and settled in the right iliac fossa. On examination he was found to be tender over the right lower abdominal quadrant, with muscle guarding. The case was diagnosed as one of acute appendicitis.

A right gridiron incision was made. The caecum was congested and oedematous, but the appendix looked normal. An attempt to withdraw a loop of small bowel failed, and a doughy mass could be felt towards the mid-line. The appendix was removed, the incision closed and a fresh paramedian incision made. The whole of the small bowel was found to be lying behind a normal-looking peritoneal layer, which extended from the transverse colon above to the pelvic brim below, and from the ascending colon and caecum on the right to the descending colon on the left. It was noted that the terminal ileum, about 2 inches from the ileo-caecal junction, passed under a narrow arch formed by this peritoneal layer. There was no opening into the sac and certainly no neck. The terminal

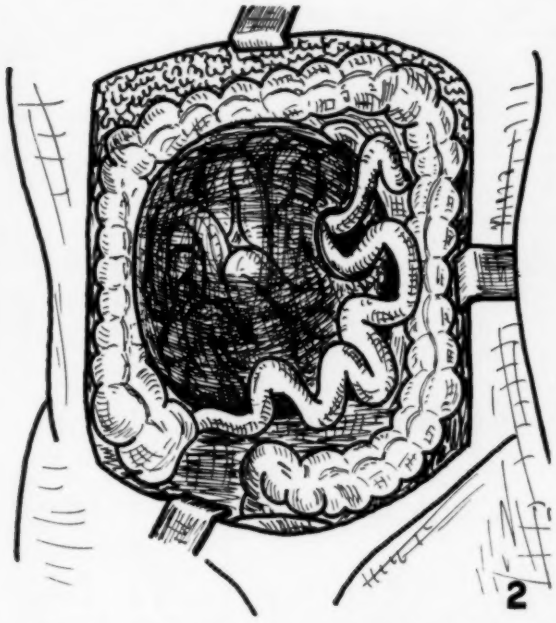

Fig. 2. Diagram of findings in case 1. A right paraduodenal-fossa hernia. Note well-formed sac with neck, and efferent and afferent loops of small bowel.

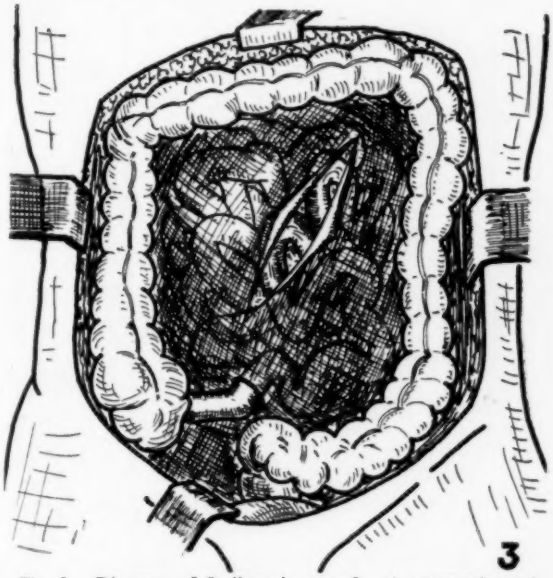

Fig. 3. Diagram of findings in case 2. A retroperitoneal hernia. The small bowel is completely covered by a translucent peritoneal layer. There is no sac and no neck.

ileum came out from the sac under the close fitting arch, but there was no entering loop. Dividing the pillars of the arch restored the normal colour of the caecum. Excision of the layer of peritoneum in most of its extent freed a normal-looking small bowel. Convalescence was uninterrupted.

This case is one of the retroperitoneal type of internal hernia. It corresponds to one of the 2 cases described by Longacre [4] in 1934: 'The small bowel was behind a glistening translucent peritoneum. The distal two-thirds of the descending colon and iliac colon were incorporated in the antero-lateral walls of the sac. Caecum and ascending colon were in the normal positions.' He points out that the significant findings in his 2 cases were the site and extent of the sac and the absence of any entering or departing loop. At no point in his case could any opening be discovered connecting the interior of the sac with the remainder of the abdominal cavity.

DISCUSSION

All authorities [2, 4, 5, 6] report on the rarity of internal hernia, and how seldom it is diagnosed pre-operatively. Lahey and Trevor [2] reported in 1945 that only 2 cases had been seen at the Lahey Clinic from 1925 to 1944, and 2 cases of right paraduodenal hernia treated surgically at the Mayo Clinic from 1910 to 1939. They reported 2 cases successfully operated upon. Reviewing the pre-operative X-rays of one case they point out that a

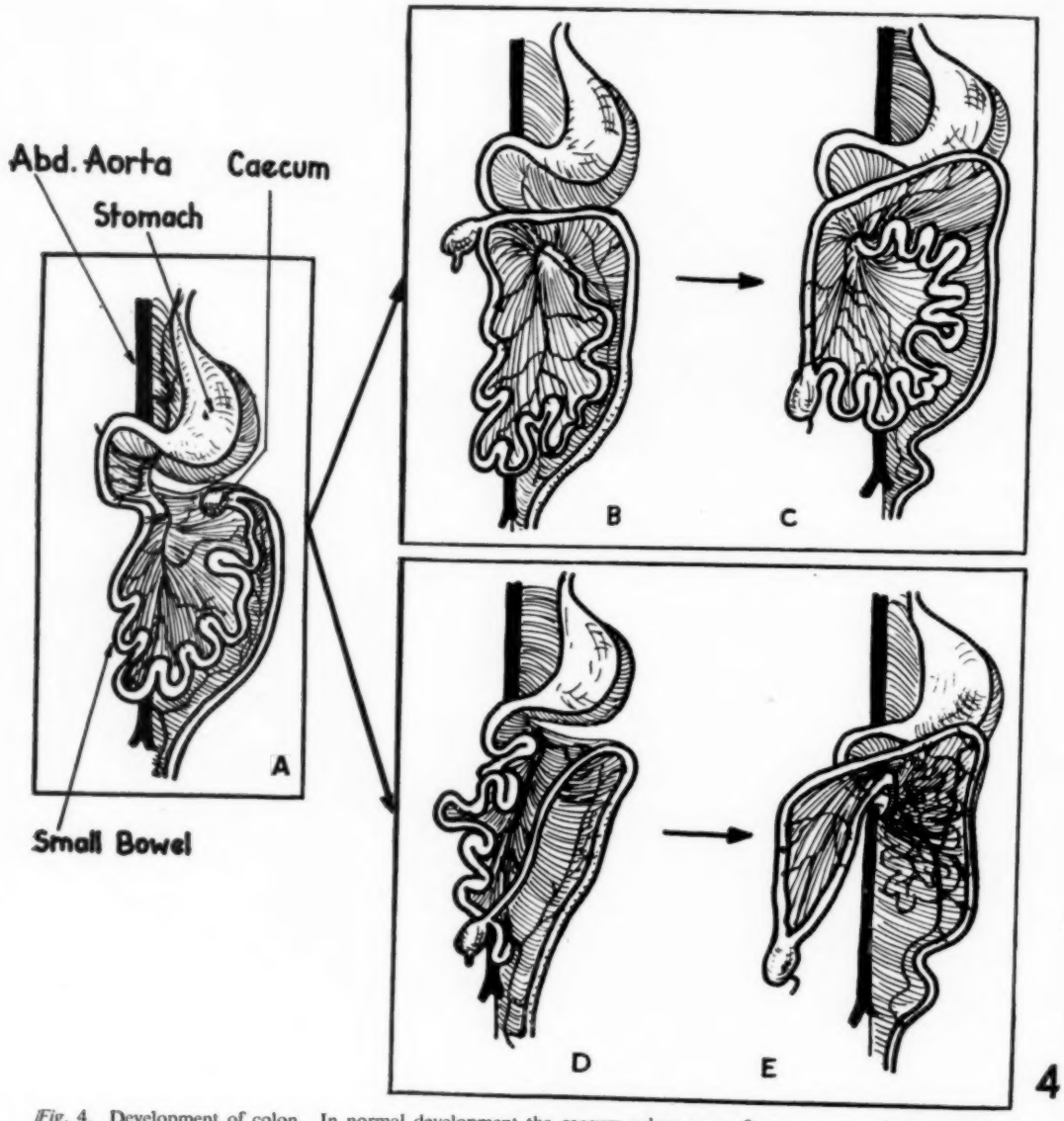

Fig. 4. Development of colon. In normal development the caecum anlage grows first across the abdomen and then down the right gutter (A, B and C). Thus it grows around the centrally placed small bowel. In abnormal development the caecum anlage grows first down then across (A, D and E). Thus it imprisons a mass of small bowel between the mesentery of the developing colon (after Andrews in *Surg. Gynec. Obstet.*[6]).

pre-operative diagnosis might have been made if the differential points described by Exner [2] were observed. These are:

1. The appearance of the small intestines as if they were contained in a spherical translucent 'paper bag', from which restricted position it is usually impossible to disturb the intestinal coils by manual palpation or postural change.

2. The location of small intestine well above the true pelvis.

Internal herniae do not as a rule produce symptoms and are usually discovered *post mortem*, at operation for another condition, or when strangulation has occurred. Few cases are diagnosed pre-operatively. Brown even states, 'So far not a single case of right paraduodenal hernia has been diagnosed before operation or necropsy.' Dowdle [7] also writes that they are rare and never diagnosed clinically.

In view of case 1, and applying the X-ray features described by Exner,[2] a pre-operative diagnosis should be possible.

The Origin of Internal Hernia

There are many divergent views as to the origin of internal hernia.

The paraduodenal type of hernial sac appears to correspond to the recesses of Landzert and Waldeyer. These recesses or paraduodenal fossae, have a large vessel in the free margin of their openings, and are named right or left according to the direction this opening faces. Treitz as far back as 1857 believed them to be the result of a widening and deepening of the paraduodenal fossa produced by pressure and peristaltic movement of small intestine. Longacre,[3] however, has shown in his dissections the paraduodenal fossae are present and empty. The case he describes is of the retroperitoneal type and the fossa would be expected to be normal.

McCarty and Present [8] report 2 similar cases to case 1. They suppose one or more loops of small intestine were caught at an early embryologic stage in a pouch formed by a very long mesentery of the proximal jejunum.

Andrews,[6] in 1923, advanced the theory that this condition was a congenital anomaly due to the imprisonment of small intestine beneath the mesentery of the developing colon. In the normal development of the colon the caecum in the upper left quadrant of the abdomen grows across the abdominal cavity, forming the transverse colon. It then passes down the right side to its normal position in the right iliac fossa (Fig.4). In doing so the developing colon passes around the small bowel, and does not imprison it. According to Andrews [6] if the mid-gut does not rotate or there is a reverse rotation, the caecum then comes to lie in the lower abdomen to the left of the mid-line. The caecum then to reach its normal position passes first down and then across the abdominal cavity, and the small bowel will be covered by the colon mesentery.

This developmental anomaly would explain the origin of a retroperitoneal hernia, but not a paraduodenal hernia with its sac and neck.

Andrews made 4 observations in his article:

1. There is no *vis a tergo* to force contents into the sac of a duodenal fossa. Differential pressures are entirely lacking in the abdomen.

2. In all but a very small minority of cases reported the degree of herniation has been total or sub-total.

3. There are hundreds of other fossae never the site of hernia.

4. The herniated viscera are never anything but small intestine.

Regarding the lack of *vis a tergo*, herniation of bowel through the foramen of Winslow does occur. As already mentioned, the duodenal fossa would be normal in the presence of the retroperitoneal type of hernia.

CONCLUSION AND SUMMARY

Two cases of internal hernia are described, of different types. The one a right paraduodenal hernia and the other a retroperitoneal hernia. It is therefore suggested that the term 'internal hernia' should include 2 types of this condition—a paraduodenal (right or left) and a retroperitoneal. The two are probably of different origin. Early authors support the older view of Moynihan, that in the para-duodenal type it is a post-foetal herniation into an unusually large pre-formed duodenal fossa. The more recent theory is that it is due to an anomaly in the development of the jejunal mesentery. The retroperitoneal type, as Andrews suggests, is an anomaly of colonic mesenteric development.

The pre-operative diagnosis seems to be possible (as in case 1), if the features described by Exner are remembered. It is important to make the diagnosis pre-operatively, for the condition is invariably fatal when strangulation has occurred. Lahey and Trevor [2] make the suggestion that in every patient with unexplained and persistent abdominal symptoms, the region of the ligament of Treitz should be explored for possibility of such a hernia.

I should like to express my thanks to Dr. K. V. O. Gunn for permission to publish his X-ray plate.

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NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

TEETHING PATTERNS IN INFANCY AS INITIAL MANIFESTATIONS OF LATENT MORBIDITIES

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This essay is an attempt to evaluate the influence, if any, which the process of teething may have upon an infant. It is based upon international literature and upon my own observations made first in general practice and later in practice as a paediatrician.

OPINIONS IN THE LITERATURE

Hippocrates (460—370 B.C.) recognized that diseases in children presented certain features quite different from those usual in adults, and he frequently referred to peculiarities of disease in children. He wrote an entire treatise on the subject of teething,[15] and thus introduced into medical thought the idea of 'Dentitio difficilis' (difficult teething) as a morbid entity, which played for centuries an important part in the diagnosis of disease—and even fatal disease—in children.

West (cited by Perabo[13]) in a statistical study of causes of death in children under 3 years of age recorded in the middle of the 19th century, found 7% 'dead from teething'. The mediaeval figure was 50%. In the years 1877—1910 this cause of death in the statistics in Berlin decreased from 1·5% to 0·3%.

Wichmann in 1797 (cited by Helmerich[8]) was the first to maintain that teething without symptoms was possible, but the belief in 'dentitio difficilis' was so strong that he met the most serious opposition and was refuted, even by clergymen, as if he had attacked a dogma.

So two extreme theories were advanced: on one side, that there are myriads of diseases and upsets due to cutting of the teeth and, on the other side, that teething is responsible for nothing but the cutting of the teeth.

It is easy to understand that many attempts have been made to compromise. Examples of this mentality are mentioned by James[9] from Anglo-American text-books (1940—1949). His conclusion is that the age-old belief that teething can make the infant ill in various ways is still valid. It may be, as he puts it, comforting to the parents and the family doctor if a frightening seizure is regarded as a transient phenomenon, unlikely to be repeated, once the dentition is completed.

On the European Continent the opposite opinion is expressed and in the teaching of medical students and—most important—of nurses too.

Pirquet, father of the term 'Allergy', and Nobel, both professors of Paediatrics in the University of Vienna, in 1928 wrote for their nurses[12]: 'The cutting of the teeth is a process without any pathological disturbances. In former times it was thought that the teething in children was very important, because all diseases with obscure etiology were attributed to the cutting of the teeth. Such an opinion is without scientific foundation.' In 1932 Pirquet's successor, Hamburger, wrote for mothers and nurses interested in paediatric nursing[7]: 'The approach of the cutting of the teeth is announced many days before by salivation and increased inclination to chew on fingers or other substances. Sometimes the children are ill-tempered and have no appetite, probably on account of sensitivity or pain in the jaws. The old, still popular, belief that teething may be the cause of more serious complaints, such as seizures, fever or diarrhoea can be a danger to the life of the child, because the doctor is not called in for early diagnosis and treatment.'

Gorter, the founder of paediatric teaching in the Netherlands and Flanders, and Hooft, who succeeded him at the University of Ghent wrote in 1938[6]: 'A nurse should know that any disease in this (teething) age has always another cause than the teething; therefore she should call a doctor.' And on page 68 of the same work: 'It is a popular belief that the cutting of the teeth is the cause of numerous discomforts and complications' This obsolete conception, only possible in those cases where the child is not well examined, disappears as soon as the doctor can diagnose gastro-intestinal disturbances, mild infections and other maladies. The mistake made is very understandable, because the period of teething is exactly the age during which the young child is most exposed to those diseases. In coincidence, cause and effect are easily confused.

Feer's opinion (1931) is most instructive for medical students and doctors[4]: '"Dentitio difficilis" does not exist in fact. It is still popular, because it is a comfort for the mother and a welcome cover for our own medical ignorance and for our complacency.' And in his text-book[5]: 'In the famous "teething-fever" teething is not the cause of the fever, but the high temperature, due to some obscure infection, activates metabolism and increases the speed of teething. Therefore this cutting of the teeth is not cause but effect.'

Among other text-books in which similar views are expressed are those of Rominger[11] (1950), Fanconi[2] (1954) and the dentist Perabo,[13] who illustrated his views with carefully investigated cases. Fanconi[2] points out that during teething many children, especially the neuropathic types, put their fingers in their mouths, causing mild infections of the gums, which are followed by a mild rise in temperature especially with thermolabile children.

THE AUTHOR'S OWN OBSERVATIONS

When I was in general practice in Java, where distances from out-lying plantations to town are vast, visits to baby-clinics by mother and child were often impossible, and for better or worse their place was taken by weighing at home, report and advice by telephone. Always the warning was given: Remember, teething is no excuse whatsoever for fits, fever, vomiting, diarrhoea, cough, restless crying, etc. If this happens, telephone and ask for advice.

First case. Telephone-call from the mother: Baby 8 months breast fed, with supplementary food; normal weight; always healthy, but not too well 10 days ago; grandmother comforted, 'Nothing but teething', and baby improved. But now again it is not too well; temperature 37·8°C (100°F). Is this teething?—No, as I told you.—Can you come? we have no transport.—After 2 hours' speeding through the hills I was at the baby's bedside, 300 feet above sea level, in malaria-infested country. After careful examination: no abnormalities detected; urine normal. But armed with microscope and a little box of stains I stained a blood-smear and examined it; after a full hour's searching—two beautiful seal rings in one erythrocyte; diagnosis—malaria falciparum. Therapy (1927) aethylcarbonas chinini (not bitter) in nicely flavoured syrup. In 4 days, long before the treatment was completed, all complaints disappeared and further teething was uneventful till the child was 3 years old, cutting the last 4 teeth of the first dentition.

The same baby produced the *second* instructive *case.* Telephone: baby is in agony, crying and screaming. No fever, no common cold, his throat is not red, he is not catching at his ears or head. He is not obstipated; on the contrary, stools are looser than usual and a little slimy. When he sits on the pot he presses, cries, presses again and refuses to stand up.—I guess I know what it is; can you come to town?—Not possible, no transport.—So I went again through the jungle. This time the diagnosis was easy, the condition had become typical: little loose stool, more mucus, with tiny blood-stained trails. A trace of this, mixed with two drops 2% eosine solution on a slide with cover-glass produced that impressive moving picture in the microscope of the amoeba histolytica 'walking' with its pseudopodium. The early diagnosis guaranteed the success of treatment.

Third case (1931). A baby 8 months old, bottle fed from 4th week, started vomiting and diarrhoea; no fever. I saw the baby the same day. In the family a common cold. Baby with acute gastro-enteritis, throat red, tonsils not swollen. Ears, lungs, urine —NAD. The therapy seemed simple; diet carefully written down. But unexpectedly the condition did not improve satisfactorily. The throat was normal after 2 days, but more loss of weight and many watery stools. Examinations again and again; glands, ears, lungs, urine, numerous bloodcounts, X-rays of the lungs—NAD. When the report from the bacteriological laboratory 120 miles away came in—No pathological organisms in stool and cultures—I was completely in the dark and asked for a second opinion. In the big town, 120 miles away, were two able specialists, a 'she' and a 'he'. She was chosen by the parents (the grandmother objected, 'Teething'). But the lady-paediatrician came and made a very good examination, first of me—I was never so cross-examined in my life—and then of the patient. She agreed that nothing in my examination was omitted and that treatment was as it should be. My proposal to take the patient with her to the Children's Hospital was found not imperative and rejected by the grandmother: 'Nonsense, it is all teething.'

But there was no improvement; after a few days more loss of weight and toxicosis was serious. As I felt uncertain about the punctual preparation of the feeding, baby was taken to the hospital, where a nurse, keen in infant feeding, nursed the child. No results. Repeated investigations—NAD. I asked for the second time for a consultant. Now 'he' had to come. And again the opinion was—therapy is right, carry on, prognosis is good. But after 10 days the baby died. A P.M. was granted—NAD, except in the mastoid antra, bilateral cloudy mucus. The consultant's opinion was that this was not the cause, but *sub finem vitae.*

At the end of the year I stopped general practice, and before I went overseas I said good-bye to the family where I lost that sweet baby. The last words of the grandmother were, 'Doctor, believe me, it was teething'! I was amazed by this comfort and resignation. I was unhappy, convinced of my ignorance.

Latent otitis (otitis without any otological or haematological symptom) as a cause of toxicosis in infants was first discovered by Ten Bokkel Huinink, Professor of Paediatrics in the University of Utrecht. He reported upon his observations and the favourable results of early operation in a meeting of the Paediatric Association in the Netherlands published in 1935[16] and 1936.[17] These observations were confirmed by Nobel in Vienna. A young doctor Liem Wie Liang, who studied at both Universities, wrote an excellent thesis[11] (1937) on this subject, so important because an early diagnosis and operation can save the life of the child.

When I returned to Java as a specialist my first difficult case was a latent otitis with toxicosis! Early operation and recovery. Demonstrating this case at a meeting of general practitioners gave me a good opportunity to stress the warning: 'No compromise! Teething is never the cause of disease.'

This resulted in a very instructive case. One of my friends, a general practitioner, asked my opinion: Child 1½ years, bottle fed, now on mixed diet, no fever, obstipated and crying more than usual, had a seizure. He examined the child carefully—NAD—and was anxious to know the cause. Teething?

We went to see the child. His examination was good, he had even tried the reflexes of Chvostek and Trousseau—negative. He rightly supposed a latent tetany, becoming manifest by hyperventilation (crying). But he did not know that these mechanical reflexes are negative during and shortly after a seizure. Now, 4 hours later, the Chvostek reflex was positive, bilateral. To make sure, I took a few drops of blood for a micro-biochemical test; the calcium in the serum, estimated in my humble lab., proved to be 8 mg. %. This confirmed the diagnosis and therapy was simple. No seizures any more, with or without teething.

A few weeks later my opinion was asked in a similar case, but it proved to be completely different. Seizures in a child 1¾ years old; obstipated. No reflex of Chvostek, serum calcium normal, but temperature 37·8°C (100°F); cell-count in the blood raised and the history—grandmother died one month ago in her home in another town of 'chronic bronchitis'. Three months ago the family and the baby had spent their holidays in the home of the old lady! Lumbar puncture: C.S.F. under pressure. Pandy positive, cell-count (30 minutes after taking) 101 (73 leucocytes, 20 lymphocytes, 8 monocytes). After 3 hours reticulum formed and stained (Ziehl Neelsen[10]) proved to contain tubercle bacilli. To find the bacilli thus is exceptional, but without this the diagnosis of meningitis tuberculosa was certain and at that time (1937) 100% fatal.

This sad state of affairs did not change before streptomycin treatment was introduced. Even in June 1948 Fanconi and Löffler[1] had to admit that more than 50% of the cases of tuberculous meningitis treated with streptomycin died. This improved when the beneficial combination with INH with or without PAS was understood and applied in specially equipped hospitals (Van Zeben[18]). Fanconi[3] (1954) has now rightly written: Results of the treatment of tuberculous meningitis will improve more and more; if treatment starts in the very beginning recovery will be nearly 100%.

This is extremely impressive because we all remember the grief and sorrow when we made that gloomy diagnosis in 1950 and before. Our helplessness has disappeared *if diagnosis is made early.*

This makes the position of the general practitioner more and more important. They should not weep. Their position is not deteriorating; it is but improving, if they stamp out the superstitions about teething, teething powders, the abuse of purgatives and all that nonsense—dangerous because delaying an early diagnosis.

In this land with its vast distances the patient and the family doctor are in more classic relationship than in any other country. Early investigation by the doctor, with his own methods *and his own microscope,* can

produce the facts necessary for early diagnosis and successful treatment at home or, if necessary, in hospital; and will gain for him the full confidence of the parents.

CONCLUSION

Teething patterns are initial manifestations of latent morbidities. They may be very valuable, because they give the first alarm to the mother, who calls the family doctor, the only man who knows how to combine family circumstances with the complaints, interpreting them by his investigations into an early diagnosis. But if she treats them as 'only teething' and fails to consult her practitioner their value as a useful warning is lost and their danger to the baby is unrepelled.

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POTENTIATED ANAESTHESIA (NEUROPLEGIA) WITH A NEW SHORT-ACTING MIXTURE CONTAINING HYDERGINE, ETAMON, PHENERGAN AND PETHIDINE

A PRELIMINARY REPORT

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The advantages (such as the prevention of shock) of carrying out operations on bad risk cases under 'potentiated anaesthesia' (neuroplegia) or hibernation represent one of the most interesting advances in anaesthesia. The principles of the method have been dealt with elsewhere.[1-3] It has been noted when neuroplegia or hibernation with the chlorpromazine (Largactil) 'lytic mixture' is applied, a more or less marked increase in pulse rate becomes manifest in most cases. To avoid this additional strain on the heart, particularly in long operations, a group of French research workers have investigated numerous drug combinations in experimental animals, and have found that Hydergine (which is a mixture of equal parts of 3 hydrogenated ergot alkaloids—dihydroergocornine, dihydroergocristine and dihydroergokryptine) in 'lytic mixtures' achieves similar effects to the original chlorpromazine mixture but does not cause tachycardia. This research group, led by J. Cahn[4], reported that their Hydergine lytic mixture gave the following results as compared with the chlorpromazine lytic mixture:

The hypothermia achieved is at least as profound and as stable, [4, 5] and affords more complete inhibition of endocrine reactions to stress.[6]

In rabbits and dogs hibernation with either the chlorpromazine or the Hydergine mixtures led to identical electro-encephalographic tracings. However, generally speaking, the electrical activity of the brain remained greater with the Hydergine mixture. Normal EEGs were re-established earlier with chlorpromazine, but on the day following the experiment no difference between the two methods could be discerned.[5, 7, 8, 9]

Hydergine had no depressant action on the myocardium as made evident by reduction of intracardiac systolic pressure and diminution of pulse pressure with chlorpromazine, and no tachycardia occurred, but on the contrary a reduction in the rate of heartbeat took place.[5, 9, 10, 11]

The two preparations, in combination with hypothermia, protected the animals equally well against the effects of ligation of a branch of the coronary artery.[5, 11]

Work carried out by Cahn *et al.*[5, 9, 10, 11] showed that good prospects for carrying out cardiac surgery without having to fear ventricular fibrillation lay in the use of a Hydergine lytic mixture in combination with infiltration of the sinus node with a local anaesthetic.

Based on the above experimental data, a number of operations on humans have been carried out with the Hydergine lytic mixture in combination with hypothermia. Huguenard[12] has reported on 17 cases of general surgery, and Campan[13, 14] on 54 neurosurgical cases. At these first clinical trials with Hydergine the following observations were reported:

The body temperature fell at the same rate as with chlorpromazine (1—1·5°C per hour) and the oxygen consumption dropped markedly. The heart rate was well controlled and tachycardia was much less frequent than with chlorpromazine. The blood pressure fell to the same extent, but the pulse pressure remained higher. By tilting the operating table controlled hypotension

could be achieved without the use of additional ganglionic blocking agents. Central sedation was less marked with Hydergine than is usual with chlorpromazine, and more anaesthetic had to be used in most cases.

PRESENT INVESTIGATION

The work referred to above was carried out by combining Hydergine with body cooling. Our endeavours were directed to carrying out 'potentiated anaesthesia' without body cooling. It was decided to try drug combinations which would not have an undesirable effect upon the heart; and because of the favourable reports referred to above, and because Hydergine has been reported to possess a protective effect against experimentally-induced ventricular arrhythmias during anaesthesia,[15-18] a drug combination containing Hydergine was selected.

Initially, Hydergine was used together with Phenergan and Pethidine. The finding of the French authors that slowing of the pulse rate occurred, was confirmed, but it was also found that an increased amount of anaesthetic drugs (Pentothal) was required. This was felt to be a drawback, and it was decided to add Etamon to this lytic mixture to potentiate its action. Etamon (tetra-ethylammonium chloride) is a potent ganglionic blocking agent. Further, it was expected that with the addition of Etamon a smaller amount of relaxing agent would be required, because of its curare-like effect.[19]

The combination of Etamon with the other ingredients of the Hydergine mixture produced the desired effect, which was not achieved when any one of the drugs was omitted.

In the course of our investigation the following combinations were tested:

(*a*) Etamon with Pethidine and the following anaesthetic drugs: Pentothal, relaxant, and nitrous oxide.

(*b*) Hydergine with Pethidine and the anaesthetic drugs mentioned in (*a*).

(*c*) Hydergine with Phenergan, Pethidine and the same anaesthetic drugs.

(*d*) Etamon with Hydergine, Phenergan, Pethidine and the same anaesthetic drugs. This final combination of 'neuroplegic drugs' (Etamon, Hydergine, Phenergan and Pethidine) produced, without addition of the anaesthetic drugs, a state of drowsiness and sleep rather resembling physiological sleep and from which the patient could readily be aroused. Furthermore, and this seems of importance, this state of 'artificial sleep' was of short duration, and could be terminated at will, as soon as the operation was concluded. Thus, a mixture allowing short-lasting neuroplegia was discovered by accident.

Technique. The following mixture was prepared:

Hydergine	(0·6 mg.)	2 ml.	} 7 ml.
Etamon	(0·2 g.)	2 ml.	
Phenergan	(50 mg.)	2 ml.	
Pethidine	(50 mg.)	1 ml.	

Of the above mixture an initial dose of 2 ml. was administered intravenously. If the patient had not fallen asleep after 10 minutes a further 2 ml. was injected. Blood pressure and pulse rate were recorded every 5 minutes (the blood pressure fell and the pulse rate, after a short initial increase, started to fall within 20 minutes from the first injection, and then remained at a stable rate throughout).

Twenty minutes after the first injection the patient was usually asleep, and at this stage 2 ml. of 5% Pentothal, and 6 mg. of curare or 50 mg. of Scoline, were given intravenously. Oral intubation was then performed, and nitrous oxide and oxygen (5 : 3) were administered. Immediately before the surgical incision a further 2 ml. of Pentothal was given in order to 'settle' the patient. During the operation, Pethidine in doses of 10 mg. was given, whenever the breathing tended to become more rapid. The re-breathing bag was squeezed repeatedly, in order to prevent carbon dioxide building up. Whenever the blood pressure rose appreciably, or the heart rate increased, a further dose of 1 ml. of the neuroplegic mixture was injected. If the patient moved, 1 ml. of 5% Pentothal was given.

The above technique was applied in 12 cases. See Table I.

Results. The general condition of the patients operated on under Hydergine-Etamon-Phenergan-Pethidine neuroplegia appeared to be good throughout. The pulse rate was reduced in all cases. The general impression gained was that Hydergine exerts a bradycardic effect in the initial stages, and in the further course of the operation,

TABLE I

Race Sex Age	*Operation*	*Duration of operation (minutes)*	*Amounts of drugs used (total)*
CM42	Appendicectomy	50	7 ml. mixture, 9 ml. Pentothal, 12 mg. curare.
EM50	Lobectomy	158	11 ml. mixture, 4 ml. Pentothal, 15 mg. curare, 60 mg. Pethidine.
BM59	Biopsy Carcinoma Tongue	17	7 ml. mixture, 25 mg. Scoline, 3 ml. Pentothal.
BM22	Skin-graft leg	38	8 ml. mixture, 6 ml. Pentothal, 3 mg. curare.
EM70	Cholecystectomy + Pancreatectomy	160	10 ml. mixture, 3 ml. Pentothal, 60 mg. Scoline, 10 mg. Pethidine.
EF58	Cholecystectomy	138	6 ml. mixture, 4 ml. Pentothal, 15 mg. curare, 10 mg. Pethidine.
BF26	Dilatation + Curettage	10	4 ml. mixture, 6 ml. Pentothal, 25 mg. Scoline.
EF63	Cholecystectomy	90	7 ml. mixture, 6 ml. Pentothal, 30 mg. Pethidine, 150 mg. Scoline, 21 mg. curare.
BM36	Bronchoscopy + Lobectomy	±180	14 ml. mixture, 7 ml. Pentothal, 125 mg. Scoline.
BF34	Dilatation + Curettage	10	3 ml. mixture, 5 ml. Pentothal.
EM73	Artificial bladder from caecum + ileum and transplant. of ureters	70	12 ml. mixture, 6 ml. Pentothal, 60 mg. Pethidine, 70 mg. Scoline, 21 mg. curare.
BM?	Intestinal obstruction	72	4 ml. mixture, 4 ml. Pentothal, 50 mg. Pethidine, 18 mg. curare.

C = Coloured; E = European; B = Bantu.
Mixture = Mixture of Hydergine, Etamon, Phenergan and Pethidine, as specified under the heading *Technique*.

does not reduce the heart rate further, but rather prevents it from rising. The pulse rate, once settled, remained almost constant throughout the operation, and never became very rapid. Usually, it was between 60 and 80, and rarely above 95 per minute.

At the end of the operation, the patient could usually be wakened easily by shouting, slapping his face gently, or moving him. If left alone he went back to sleep immediately. The action of the neuroplegic mixture was short-lived, no matter how long the operation had lasted. Nevertheless the patients required little or no sedation in the first few post-operative hours. Throughout the operation, the skin was dry, cool and pink. In some cases sweat appeared, and the administration of 10 mg. of Pethidine terminated this reaction.

The body temperature usually dropped by 3—4°F. (experiments on white mice with this mixture resulted in a drop of temperature of 7—9°F over a period of ½ to ¾ hours.)

It was observed when this neuroplegic mixture was used that much less relaxant had to be used than was otherwise expected. Some cases could be intubated with as little as 3 mg. of curare and 1 ml. of Pentothal.

The amounts of neuroplegic mixture varied from case to case. One patient who had received only 4 ml. of neuroplegic mixture was asleep and analgesic for about ½ hour.

A point of particular importance is the remarkably good post-operative condition of the patients, on which the house surgeons commented.

SUMMARY

1. This is a preliminary report on the use of a new short-acting neuroplegic mixture for use in 'potentiated anaesthesia' (neuroplegia).

2. The neuroplegic mixture consisted of Hydergine, Etamon, Phenergan and Pethidine.

3. Anaesthesia was maintained with Pentothal, nitrous oxide, Pethidine, and relaxants.

4. In all cases the pulse rate was reduced.

5. The amount of relaxant required was markedly reduced.

6. The state of neuroplegia produced was of short duration, lasting as long as the patient was given just sufficient anaesthesia to keep him asleep.

7. This method seems to afford good protection from shock, as evidenced by a dry, cool and pink skin throughout operation, and a remarkably good post-operative course.

I wish to express my thanks to Dr. P. Toker (Chief Anaesthetist), Mr. A. E. Laubscher (Head of the Department of Surgery and Mr. B. M. de Saxe (Surgeon, Boksburg-Benoni Hospital) for their forbearance and encouragement. The supplies of Hydergine required for this investigation were made available *gratis* by the manufacturers, Sandoz Ltd., Basle, Switzerland, and are acknowledged with thanks.

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ATROPINE IN MUSHROOMS

THERAPEUTIC IMPLICATIONS

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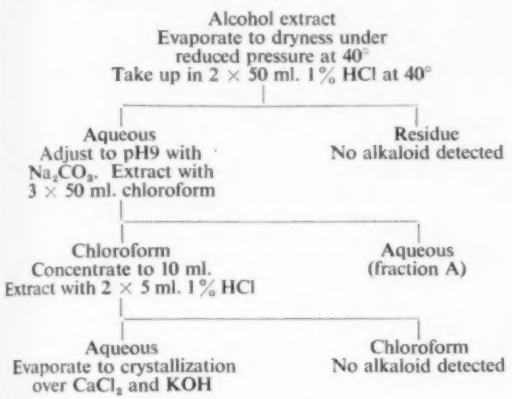
The fungi *Amanita muscaria* and *A. pantherina* are the commonest toxic species in the Western Cape, possibly in the whole of South Africa.[1] Treatment of poisoning by these varieties is usually based on the assumption that muscarine is the chief poison involved. However, attention has frequently been drawn to the fact that the neurological signs, confusion delirium, convulsions and dilatation of the pupil are inexplicable on this hypothesis; on the other hand they resemble the effects of atropine overdosage.[2, 3, 4] Kobert invoked an unidentified tropine pharmacologically resembling atropine, which he termed 'pilzatropin', while de la Rivière referred to 'myceto-atropine'. According to Ainsworth [5] nervous symptoms 'are generally attributed to muscaridine'.

The therapeutic significance of this atropine-like substance was emphasized recently;[4] the administration of atropine to antagonize symptoms due to so-called muscarine poisoning would be inadvisable if neurological signs due to 'myceto-atropine' were predominant.

PRESENT INVESTIGATION

In an attempt to characterize the alkaloids 1·0 kg. each of *A. muscaria* and *A. pantherina* were gathered in the

pine woods in the vicinity of the University of Cape Town during the early winter. After being dried at 60° they were extracted with 96% alcohol in the Soxhlet apparatus for 72 hours, and subsequently worked up according to the following scheme:



The crystalline yield from *A. muscaria* was 1·1 mg., and from *A. pantherina* 1·8 mg. In both cases reactions were obtained with several alkaloidal reagents; however, ferricyanide was not reduced, as would be expected of a betaine such as muscarine. The properties are compared in Table I with l-hyoscyamine. The material in both

TABLE I

	A. pantherina	*A. muscaria*	l-*Hyoscyamine*[8]
Melting point	107·5°	108°	108·5°
Crystalline form	Colourless needles	Colourless needles	Silky needles
Melting point of aurichloride	164°	163°	165°
Rf	0·7	0·7	0·72
Vitali's test	Positive	Positive	Positive
Bio-assay (rabbit pupil dilatation)	1·0 microgram active	Not tested	±0.5 microgram

cases was studied by paper chromatography with the solvent n-butanol 86 parts with 14 of acetic acid, saturated with water. The spots were developed with iodine vapour. The yields appeared homogeneous, Rf 0·7.

Fraction A from both species gave the reactions of a betaine, and probably contained muscarine.

Discussion

The alkaloids obtained from *A. muscaria* and *A. pantherina* were not distinguishable from l-hyoscyamine by the tests performed. This substance is the commonest naturally-occurring alkaloid of the tropane group.[6] While not ignoring the occurrence of geographical variation in alkaloidal content of mushrooms,[7] it appears that Kobert's 'pilzatropin' is in fact l-hyoscyamine, and that the neurological signs due to poisoning by mushrooms of the species studied are due to this alkaloid.

Atropine is the racemic (dl) form of l-hyoscyamine, and in its peripheral effects possesses about half the activity of the latter.

It has therefore been confirmed that atropine should not be used indiscriminately in the treatment of poisoning by *A. muscaria* and *A. pantherina;* the relative severity of atropine-like and muscarine-like effects should be assessed, and appropriate therapy administered.

SUMMARY

Two poisonous mushrooms abundant in the Cape, *Amanita muscaria* and *A. pantherina*, have been studied chemically and a substance with the properties of l-hyoscyamine isolated.

Certain of the features of poisoning produced by these fungi are probably due, not to muscarine, but to l-hyoscyamine. This should be borne in mind when assessing cases for treatment, as administration of atropine would aggravate the degree of poisoning.

Sincere thanks are due to Dr. N. Sapeika, Miss E. L. Stephens, and Mr. H. Williams for advice, and to Professor W. Pugh who provided facilities for this investigation.

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QUESTIONNAIRE ON REGISTER OF SPECIALISTS

FURTHER REPORT BY SCRUTINEERS

A further report[1] by the four Scrutineers in the recent questionnaire on the Register of Specialists has been addressed to the Secretary of the Medical Association of South Africa as follows:

Your letters of 15 November and 31 December 1954 refer.

We have given this matter some thought and have come to the following conclusions:

1. That the task for which we, as scrutineers, were appointed has been adequately fulfilled and reported upon in the *South African Medical Journal.*

2. The further information to be extracted from the voting papers in order to determine

(*a*) 'How many votes were recorded by Specialists and General Practitioners and that this should be further sub-divided into those practising in urban areas and those practising in rural areas' (your letter of 15 November 1954)

(*b*) 'The Council desired to be advised as to how the votes were divided between Specialists and General Practitioners, and, if possible, how the votes were recorded by those living in the larger centres and those in the country areas' (your letter of 31 December 1954)

appears to us to be well-nigh impossible without a clearly defined list of the registered medical practitioners who voted, classified

as 'those practising in urban areas and those practising in the rural areas' or by 'those living in the larger centres and those in the country areas'.

We are prepared to attempt the task set us by Federal Council, provided the Executive of Federal Council would clearly define which centres in the Union are to be regarded as urban and which centres are to be regarded as rural in terms of the resolution of Federal Council.

The reply to this is contained in your letter of 21 January 1955, as follows:

With further reference to your letter of the 8th instant, I would advise that in the opinion of the Executive Committee of Federal Council it will be sufficient for the scrutineers to proceed with the classification of the results of the questionnaire into Specialists and General Practitioners, and to deal with the other question regarding area of practice, i.e. rural or urban, on broad lines. One member has even suggested that these areas have been defined by certain Control Boards, e.g. the Meat Control Board. Others have referred to the larger centres being taken as urban areas. For example, Dr. Maurice Shapiro writes: 'The definition of "urban" and "rural" presents a real difficulty. I suggest that a classification based on the defined areas of Branches and Divisions of the Association would meet the position. Alternatively, Johannesburg, Pretoria, Bloemfontein, Kimberley, East London, Port Elizabeth, Durban and Pietermaritzburg could be taken as the "larger centres" and the rest could be lumped together as "country areas".'

There was thus no clear directive. We have used our own discretion in trying to fulfil the task which Federal Council had set us.

We scrutinised each individual voting paper and with the aid of the latest register of the S.A. Medical and Dental Council we classified each into two main groups, viz. the Specialists and General Practitioners. The General Practitioners were then further classified into the two groups, viz. the urban General Practitioners practising in the large urban centres, and the rest were looked upon as practising in the smaller centres or country General Practitioners.

A fourth class of medical practitioners was established as 'Others' and these included full-time medical officers, interns and those who were overseas at the time of voting.

The counting of the votes, according to the A, B, C, D sections of the questionnaire, was undertaken by the scrutineers originally appointed by Federal Council, assisted by a few volunteer helpers.

Whilst the totals of the counts are not 100% correct, they are reasonably accurate to give reliable figures of those votes cast within the categories established by the scrutineers.

The results obtained are set out below.

Number of voting papers dealt with: 2,597.

Under Section A—Are you in favour of a reversion to the system which existed prior to the introduction of the Specialist Register in 1938?

the votes were	Yes 292 No 2,305
Urban General Practitioners ..	996
Country General Practitioners	756
Specialists	575
Others (full-time medical practitioners, interns, medical practitioners overseas) ..	270

The 2,305 who did not vote Yes under Section A cast their votes under Sections B, C, D as follows:

The 2,305 votes were made up as follows:

Urban General Practitioners ..	860
Country General Practitioners	689
Specialists	532
Others	224

	Urban GPs	*Country GPs*	*Specialists*	*Others*
Section B				
1. Are you in favour of a Register of Specialists only?	119	102	331	59
2. Are you in favour of a Register of Consultants only?	619	450	71	106
3. Are you in favour of a Register of Specialists plus a Register of Consultants?	110	127	115	57
Indefinite answers ..	12	10	14	5
Section C				
Are you in favour of a Statutory Register?	516	368	468	162
Are you in favour of a Voluntary Register?	334	289	51	59
Indefinite answers ..	10	28	13	3
Section D				
Do you think that Specialists should be allowed to do domiciliary visiting? Yes	41	32	62	20
No	802	648	459	194
Indefinite answers	17	7	11	10

Signed by Scrutineers

J. P. de Villiers
J. A. Currie
A. I. Goldberg
J. R. E. Lee

28 February 1955

1. The first report of the Scrutineers was published in the *Journal* of 18 September 1954 (**28,** 815).

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Ivor Lang, of Port Elizabeth, who was in charge of the cinematograph arrangements for the 1954 Medical Congress, has been elected President of the Port Elizabeth Film Group. He has just been awarded the Stanley Nathan and Hillary Wood floating trophies for the best film of the year, entitled 'Land of Nectar'.

* * *

Cape Western Branch of the Radiological Society of South Africa. At a meeting held on 28 February 1955 the following were elected office-bearers for 1955: *Chairman*—Dr. James Latham, *Vice-Chairman*—Dr. Adolph Meyer, *Hon. Secretary*—Dr. Kenneth Brauer.

* * *

Dr. B. Th. Kranz (residence 371 Main Street, Paarl) has joined Dr. B. M. Kranz (residence Bellevue Street) in partnership. Consulting rooms: 373 Main Street, Paarl. Telephone: Paarl 1111.

Dr. L. J. Abramowitz, M.B., Ch.B. (Cape Town), M.R.C.O.G., has joined Dr. D. Friedlander, M.B., Ch.B. (Cape Town), L.M., M.R.C.O.G., in his practice as a specialist in Obstetrics and Gynaecology at 605 Diamond House, Parliament Street, Cape Town.

* * *

The Author of the article *Michael Servetus His Importance in the History of Medicine* which was published in this *Journal* on 26 February 1955 (**29**, 208), and the Editor, desire to acknowledge the source of the passages quoted from Professor C.D. O'Malley's book *Michael Servetus, a Translation of his Geographical, Medical and Astrological Writings with Introductions and Notes* which was published by Messrs. Lloyd-Luke (Medical Books) Ltd., of London.

Medical Training School for the University of Stellenbosch. A further step towards the establishment of a general hospital in the Parow-Goodwood area, to serve as a medical training school for the Medical Faculty of Stellenbosch University, has been taken by the Cape Provincial Administration by the advertising of the post of Medical Superintendent of the proposed hospital in current issues of this *Journal*. It is understood that the incumbent of the post will be enabled to collaborate in the planning and design of the hospital and will be afforded every opportunity to become *au fait* with all aspects of hospital administration prior to its opening.

AMERICAN SPECIALIST TO INVESTIGATE HEART DISEASE IN THE UNION

Dr. Ancel Keys, Director of the Institute of Physiological Hygiene of the University of Minnesota U.S.A. has arrvied in Cape Town to study the incidence of heart disease in South Africa and to collaborate with research workers here. Dr. Keys's visit which will last until 1 April, was arranged through the South African Council for Scientific and Industrial Research, and during his stay he will work with the C.S.I.R.-University of Cape Town Research Unit in Clinical Nutrition (Director Prof. J. F. Brock), and will lecture to medical men, insurance executives and to a meeting of the Council for Scientific and Industrial Research.

Dr. Keys is a world authority on food in relation to heart disease and his researches here will be conducted among three racial groups living in Cape Town. It was known, he said, that all over the world the incidence of the disease was lower in certain populations or segments of populations than in others, and it was believed that the dietary factor was an important, if not the most important, factor in heart disease.

In Cape Town the theory that race affected the incidence of the disease would be tested. He did not think that race would prove important in the fight against the disease. South Africa, and Cape Town in particular, was unique because there were three different racial groups, each with a stable population with different habits and diets, but all subject to the same climatic conditions and with similar stresses in their daily lives.

POLIOMYELITIS IN THE UNION

Following are the returns, supplied by the Union Department of Health, of cases notified under the Public Health Act as suffering from Poliomyelitis in the period 17 to 24 February 1955.

	European	*Non-European*
Transvaal:		
Johannesburg	5	2
Johannesburg P.U.A.H.B.	1	
Germiston	2	
Vanderbijlpark	1	
Pretoria	1	1
Pretoria P.U.A.H.B.	1	
Lyttelton	1	
Vereeniging	1	
Randfontein P.U.A.H.B.	1	
Benoni	1	
Benoni P.U.A.H.B.	1	
Middelburg	2	
Total for Transvaal	18	3
Cape Province:		
Cape Town Municipality		1
Matatiele district		1
Vredendal Village Management Board		1
Port Elizabeth	1	
Oudtshoorn Divisional Council		1
Wellington Municipality		1
Total for Cape Province	1	5
Natal:		
Durban	6	1
Pietermaritzburg	3	
Edendale		1
Kloof	1	
Bergville		1
Mapumulo district		1
Ixopo district		1
Glencoe	1	
Stanger district		1
Ndwedwe district		1
Total for Natal	11	7
Orange Free State:		
Bloemfontein	2	
Kimberley		1
Total for O.F.S.	2	1
TOTAL FOR THE UNION	**32**	**16**

Union Department of Health Bulletin. Report for the 7 days ended 24 February 1955:

Plague. Cape Province: Three (3) Native cases in the St. Marks district. Diagnosis based on clinical grounds only. Laboratory confirmation awaited.

Smallpox: Nil.

Typhus Fever. Cape Province: One (1) Native case in the Herschel district. Diagnosis confirmed by laboratory tests. *Orange Free State:* No further cases have been reported from the Koppies area since the notification of 27 January, 1955. This area is now regarded as free from infection. No further cases have been reported from the Harrismith district since the notification of 27 January, 1955. This area is now regarded as free from infection.

Epidemic Diseases in other Countries:

Plague: Nil.

Cholera in Dacca, Chalna (Pakistan).

Smallpox in Kabul (Afghanistan); Moulmein, Rangoon (Burma); Phnom-Penh (Cambodia); Allahabad, Bombay, Calcutta, Delhi, Kanpur, Lucknow, Madras, Nagpur, Tellicherry (India); Karachi (Pakistan); Nhatrang, Phanthiet, Saigon-Cholon (Viet-Nam); Mogadiscio (Somalia).

Typhus Fever in Kabul (Afghanistan).

BOOK REVIEWS : BOEKRESENSIES

KWASHIORKOR

Kwashiorkor. By H. C. Trowell, *O.B.E.*, M.D., F.R.C.P., J. N. P. Davies, M.D. and R. F. A. Dean, Ph.D., M.R.C.P. (Pp. 308 + xii, with illustrations. 50s.) London: Edward Arnold (Publishers) Ltd. 1954.

Contents: Part I. Reports of Kwashiorkor in Children and a Discussion of Terminology. *Part II.* The History of Kwashiorkor. *Part III.* Kwashiorkor in Children. *Part IV.* Protein Malnutrition in Adults. *Part V.* Implications of Kwashiorkor in Children and of Protein Malnutrition in Adults.

This monograph is to be very warmly welcomed as the first full and detailed account of this interesting disease. The senior author has been a pioneer in the study of kwashiorkor in East and Central Africa. Dr. Davies as Professor of Pathology at Makerere University in Uganda (the inter-territorial University of British East

Africa) has made an intensive and interesting study of the pathology of Uganda Africans and especially of kwashiorkor. Dr. Dean after extensive experience of the correction of severe undernutrition in children in Germany in the post-war period has been appointed Director of a group for the study of infantile malnutrition in Uganda and is financed by the Medical Research Council of Great Britain. These three have combined in an authoritative account of kwashiorkor in Uganda which must be a standard reference not only for Uganda but for Africa and also for related syndromes in other parts of the world which are increasingly coming to be called kwashiorkor. Many of the alternative names coined in other parts of the world are given in the monograph and a more complete list is given as an appendix to the report of the third session of the Joint FAO/WHO Expert Committee on nutrition (FAO Nutrition Meetings report No. 7, Rome, December, 1953).

The monograph accepts the main recommendations of the third session of the Joint FAO/WHO Expert Committee in Nutrition which met at Fajara, Gambia, in November 1952, following an African Conference on this subject organized by C.C.T.A. In general definition and nomenclature kwashiorkor is regarded as a dietary disease resulting from protein malnutrition. Non-dietary factors may contribute but the disease does not develop when diet is reasonably satisfactory in respect of proteins. Protein malnutrition is defined by the third session of the Joint Committee. It is rather a pity that this definition has not been reproduced in the monograph since there have been many misunderstandings of the term 'Protein Malnutrition'. As used by the Joint Committee it refers to deficiency of protein foodstuffs, not of protein as a pure combination of amino acids. Protein malnutrition is best seen where calories are generously provided by starchy foodstuffs but, where both calories and protein foodstuffs are deficient in the diet, varying clinical pictures form a transition from typical kwashiorkor to undernutrition and marasmus. To attribute kwashiorkor to dietary protein malnutrition is not equivalent to calling it protein deficiency in the sense of a pure combination of amino acids. Protein malnutrition as defined by the Joint Committee allows a role in causation not only for amino acids but also for vitamins, known and unknown, and even trace mineral elements. The extent to which amino-acid deficiency is the limiting factor in the production of the deficiency state has not yet been settled.

There are many people, including some workers in South Africa, who regret the adoption and popularization of the term 'kwashiorkor'. To a limited extent the reviewer shares this feeling, but recognizes that the Joint Committee was faced with the problem that it could not find a better name among the long list in the appendix to the third report. Objection to the use of the term 'kwashiorkor' has probably been most clearly expressed by Gomez *et al.* from Mexico City (Acta paediatrica, 1954, **43**, 336). The plea of these workers is for the use of the term 'malnutrition' (desnutricion) in preference to all other names. They propose to classify 'malnutrition' simply by degrees—first, second and third. With this opinion the reviewer cannot agree, and the general use of the term 'kwashiorkor' seems to be the lesser of two evils.

There has been a general international tendency recently to confine the term 'kwashiorkor' to protein malnutrition as seen in the breast-feeding or post-weaning child. This tendency seems to follow on the etymological conclusion that kwashiorkor means 'the deprived child'. The authors of this monograph, however, point out that protein malnutrition with indistinguishable clinical features occurs also in adolescents and adults, and they have a section of their book devoted to 'protein malnutrition in adults', in which they recommend the label kwashiorkor.

Altogether this is a very fine piece of work, which will constitute an important reference for the study of protein malnutrition throughout the world. It is also a monument to the British Colonial Medical Service in Africa and to the Mulago Medical School at Makerere University.

J.F.B.

FAT METABOLISM

Fat Metabolism. By Victor A. Najjar. (Pp. 185 + viii, with illustrations). Baltimore: The Johns Hopkins Press. 1954.

Contents: 1. Clinical and Biochemical Features of Fat Metabolism. 2. Obesity in Childhood—Some Clinical Aspects. 3. Multiple Causative Factors in Obesity. 3a. Discussion: Constitutional Factors in Obesity. 3b. Discussion: Endocrine Factors in Obesity. 4. Lipemia. 4a. Discussion Essential Hyperlipemia. 5. Preparation, Utilization, and Importance of Neutral Fat Emulsion in Intravenous Alimentation. 6. On the Role of Lipemia Clearing Factor in Lipid Transport. 7. Consideration of the Role of Coenzyme A in Some Phases of Fat Metabolism. 8. Enzymatic Oxidation and Synthesis of Fatty Acids. 8a. Discussion: A Contribution to the Mechanism of Diabetes Mellitus. 9. Lipogenesis *in vitro* and its Hormonal Control. 10. Lipid and Phospholipid Synthesis. 11. Some Aspects of Cholestrol Metabolism Related to Atherosclerosis. Index.

This book is a companion volume to the symposium on carbohydrate metabolism by the same editor which was published in 1952 and reviewed in this journal.

Fat metabolism has been the subject of many hypotheses; β-oxidation, multiple alternate oxidation, and acetic-acid condensation have in turn held the field. In the last 5 years many of these problems have been solved by the discovery of active acetate, a two-carbon-fragment activated combination with coenzyme A. Active acetate appears as the essential stepping-stone in the synthesis and oxidation of fat. By its first condensation active acetate forms acetoacetate-coenzyme A, an activated form of acetoacetate; this is the first step in building up the fatty-acid chain and also provides an important normal metabolite. Active acetoacetate is unstable and when produced in excess by over-active fat-metabolism much of it may lose its coenzyme A and with it its reactivity. Plain acetoacetate is left, which is disposed of with difficulty and is the cause of ketosis.

Active acetate may enter the citric-acid cycle and may also be derived from carbohydrate metabolism, so that one link between fat and carbohydrate metabolism has become clear. Active acetate is also the essential building brick in the synthesis of cholesterol, which lies under such heavy suspicion in connection with the causation of atherosclerosis.

This book gives an account of this progress in biochemistry. There are also essays on the psychosomatic approach to the problem of obesity; on lipaemia, heparin and the clearing factor; and on fat emulsions for intravenous feeding.

G.C.L.

ADVANCES IN ENDOCRINOLOGY

Recent Advances in Endocrinology. P. M. F. Bishop, D.M. (Oxon.), M.R.C.P. (Lond.). Seventh edition. (Pp. 348 + viii, with 34 illustrations. 30s.) London: J. & A. Churchill, Limited. 1954.

Contents: 1. The Adrenal Cortex. 2. The Endocrine Control of Carbohydrate Metabolism. 3. Cushing's Syndrome. 4. Hermaphroditism, Pseudohermaphroditism and Macrogenitosomia Precox. 5. Precocious Puberty and Sexual Precocity. 6. Testicular Deficiency. 7. Virilism and Hirsutism. 8. Clinical Use of Oestrogens. 9. Hormones and Cancer. 10. The Management of Thyrotoxicosis. Appendix I and II. Index.

Recent Advances in Endocrinology was first published by the late Professor A. T. Cameron in the year 1933, and had reached the 6th edition at the time of his death in 1947. Seven years had, therefore, elapsed before the appearance of the present edition.

The publishers have been very wise in their choice of Dr. P. M. Bishop as author of this edition. He has achieved fame as an endocrinologist and is consultant to Guy's Hospital, Chelsea Hospital for Women and the Post-Graduate Medical School of London.

This book has been completely recast and rewritten. When Professor Cameron first wrote the book in 1933, there were practical lyno text-books on the subject written in the English language and he performed a valuable service in covering the whole ground. Endocrinology has advanced at a prodigious rate and there are now many excellent text-books. Dr. Bishop has, therefore, chosen to deal with only certain aspects of endocrinology. He has also viewed the subject from a different angle, as Professor Cameron was a biochemist and Dr. Bishop is a clinical endocrinologist.

The chapter on the adrenal glands contains a lucid description of the chemistry and actions of the steroids. In addition to clinical syndromes, there is a practical discussion of the use of ACTH and cortisone.

Recent work on the various aspects of carbohydrate metabolism is adequately presented and the endocrine factors are stressed.

Dr. Bishop has had an intensive experience with Cushing's syndrome and this chapter is most informative. In addition, his vast experience in the treatment of sex disorders in the female is summarized in a most valuable chapter.

The management of the thyrotoxic patient is of great concern to every practitioner. The author has presented an unbiassed assessment of the different treatments which should facilitate one's approach to the problem.

Precocious puberty, sexual precocity, pseudo-hermaphroditism, testicular deficiency, virilism, hirsutism and the place of the hormones in cancer, are dealt with in a clear and instructive manner.

All the important advances are well covered and the book contains a wealth of information. It should be read with great benefit by the general practitioner, physician and endocrinologist.

Dr. Bishop is to be congratulated for the manner in which he has dealt with a most difficult task and it is hoped that he will continue to edit this book for many years to come.

S.L.

LEG ULCERS

Leg Ulcers. Their Causes and Treatment. By S. T. Anning, T.D., M.A., M.D. (Cantab.), M.R.C.P. (Pp. 178 + viii, with 42 illustrations. 18s.) London: J. & A. Churchill, Ltd.

Contents: 1. Historical Survey. 2. The Normal Circulation of the Lower Limb. 3. The Causes and Results of Failure of the Leg Muscle Pump. 4. Clinical Conditions causing Venous Thrombosis. 5. The Pathology of Venous Thrombosis. 6. Arterial Disease and Leg Ulcers. 7. The Influence of Heredity. 8. Miscellaneous Factors. 9. Treatment. Conclusion. References. Author Index. Subject Index.

This monograph is little more than a review of the literature and the 1026 cases seen by the author. There is no evidence of basic investigations having been done to solve the real problems of this condition. One thus finds that, although virtually the whole book is devoted to venous ulceration, there are no anatomical dissections, venous pressure readings or phlebograms illustrated (but there are numerous illustrations of how to bandage the ulcerated leg).

There are some interesting observations regarding heredity, hypertension, obesity, occupation and the relative incidence of the various types of leg ulcers, particularly the fact that 4·1% were the direct result of the injection of varicose veins.

The author is a dermatologist; so one does not expect surgical management to be stressed but, even though surgery has a limited place in the treatment, it warrants more than the 5 pages allocated to it.

The list of references is large but it is surprising to see that Sherman's work has been missed out. Its inclusion will be a decided improvement.

D. J. du P.

BRITISH ENCYCLOPAEDIA OF MEDICAL PRACTICE: 1954

The British Encyclopaedia of Medical Practice: Medical Progress 1954. Edited by Lord Horder, G.C.V.O., M.D., F.R.C.P. (Pp. 281 + vi). London: Butterworth & Co. (Publishers) Limited. South African office: Butterworth & Co. (Africa) Ltd., Durban. 1954.

Contents: Part I. Critical Surveys. 1. Medicine. 2. Surgery. 3. Obstetrics and Gynaecology. 4. Chest Surgery. 5. Neurology. 6. Gastro-enterology. 7. Psychological Medicine. 8. Occupational Skin Diseases. 9. Progress in Forensic Medicine. 10. Physical Medicine. 11. School and Priority Dental Services.
Part II. Drugs. 12. Recent Developments in Pharmacology and Therapeutics.
Part III. Abstracts. 13. Abdominal Emergencies—Whooping Cough. Index.

This book is in 3 parts—the first consists of critical surveys covering nearly the whole field of medicine in 11 chapters, each writer being an expert in his particular field.

The Editor-in-Chief—Lord Horder—needs no introduction. In his foreword he quite rightly and with full justification remarks: 'Medical aspects of cardiac, vascular and peripheral arterial disease are claiming a large amount of interest, and in these conditions there is an excellent example of team work—the physician, surgeon and technician all playing their part'. In the chapters dealing with these conditions the reader will find the most recent work and results of the British School set out. There is no doubt of the enormous interest of all sides of the profession in chest and cardiac surgery.

The chapter on Physical Medicine by Francis Bach needs special mention. He is a dynamic writer and sets out as his object the attaining and retaining of positive health. He describes two trends: first the development and use of instruments of precision, and secondly the organization and coordination, particularly in the hospital, of rehabilitation. The future of this branch of medicine is exciting and assured.

In Part II A. D. Stuart deals with recent developments in Pharmacology and Therapeutics; and to the general practitioner, this is indeed a valuable addition to this volume.

Part III consists of abstracts from publications in nearly every country where medicine is practised, and fills over 100 pages of the book, covering the entire field of medicine. A bibliography and full references are provided.

I can recommend this volume wholeheartedly—the contents represent the best of the British School of Medicine. The printing and binding are excellent. One criticism: why cram the first and last pages with advertisements of drugs and books? Most practitioners get an overdose of proprietory advertisements as it is, and one can at least expect a book of this calibre to refrain.

A.M.M.

MEDICINE AT THE VIENNA UNIVERSITY

The Medical Faculty of the Vienna University. By Prof. Dr. Fritz Driak. (Pp. 71, with 17 illustrations). Austria: Urban & Schwarzenberg, 1954.

Contents: 1. From the History of the Medical Faculty of the Vienna University. 2. The Medical Faculty of the Vienna University in Modern Times. 3. The Study of Medicine at the Medical Faculty of Vienna University in Modern Times. 4. Post-graduate Training of Medical Doctors in Austria. 5. The Life of the Student in Vienna—Concluding Remarks.

This small book of 71 pages has been prepared by Professor Driak as a guide to Austrian students and students from foreign countries who study at the Medical School of the Vienna University.

The students cannot fail to be impressed by the history of the Alma Mater Rudolphino Vindobonensis whose records in the Medical Deanery have been preserved without interruption since 1399, the oldest documents of the hospital dating back to the year 1200. And what a formidable array of names record the glories of the past!

Then follows an outline of the programme of study for the undergraduate and the post-graduate students with a comprehensive list of facilities available to them. After five years the successful student receives a diploma of doctor of medicine but is not allowed to enter private practice until he has served a further three years in a public hospital or other approved institution under guidance and supervision of the head of the institution. Only then is he permitted to practise the medical profession independently and entitled to call himself a 'practitioner'.

A.K.

CLINICAL ENDOCRINOLOGY

Clinical Endocrinology. By Laurence Martin, M.D., F.R.C.P. and Martin Hynes, M.D., M.R.C.P. Second Edition. (Pp. 253 + ix with 39 illustrations. 20s.) London: J. & A. Churchill, Ltd. 1954.

Contents: 1. The Pituitary. 2. Fröhlich's Syndrome and Obesity. 3. The Pineal Body. 4. The Thyroid Gland. 5. The Parathyroid Glands. 6. The Thymus. 7. The Adrenal Glands. 8. The Testes. 9. The Ovary. 10. The Breast. 11. Hormone Implantation.

The second edition of this book is to be warmly welcomed after the passage of 6 years. There are many recent large volumes and reference works on endocrinology, and they must be very confusing to the student and general practitioner. There is need for a simple clearly-set-out description of the principal endocrine abnormalities and their differentiation and treatment. This need, as Sir Lionel Whitby says in his foreword, is admirably met by this book. In the preface to the second edition the authors say, 'In order to keep within the bounds of our subject, and to avoid embarking upon a textbook of general medicine, we have not given accounts of the numerous non-endocrine diseases for which cortisone or ACTH can be used in treatment'.

The book is fully up to date in such matters as aldosterone and the treatment of Addison's disease, but it is surprising to find no reference to adrenalectomy in the index. From the local African point of view it is surprising to see the subject of gynecomastia dealt with without reference to malnutrition.

The subject of the development of nodules in simple goitre is dealt with in a most exemplary way, even if there are still some who may not agree with the views expressed. In the reviewer's opinion all that is needed to complete the account on pages 64 and 65 is a statement that there is no such thing as a chronic diffuse goitre without nodules.

The language is lucid, the clinical description exact, and the recommendations for treatment concise and conservative. No general practitioner or student could read this book without profit and pleasure; and there are few physicians to whom the same remarks would not apply.

J.F.B.

BOOKS RECEIVED : BOEKE ONTVANG

A Further Study in the Nature of Diseases. By J. E. R. McDonagh, F.R.C.S. Pp. 372. 21s. London: William Heinemann Medical Books Ltd. 1955.

Acta Endocrinologica. Edited by Axel Westman. Volume XVII. Pp. 460 with illustrations and tables. Copenhagen: Ejnar Munksgaard. 1954.

A Therapeutic Index. By C. M. Miller, M.D. (Lond.), M.R.C.P. (Lond.) and B. K. Ellenbogen, M.D. (L'pool), M.R.C.P. (Lond.). First edition. Pp. 147. 12s. 6d. London: Baillière, Tindall and Cox. 1955.

Reflections on Renal Function. By James R. Robinson, M.D., Ph.D., (Cantab.). Pp. 163. 17s. 6d. Oxford: Blackwell Scientific Publications. 1954.

The Development of Tropical and Sub-Tropical Countries. Edited by A. Leslie Banks, M.A., M.D., F.R.C.P., D.P.H. Pp. 217 + xvi. 18s. London: Edward Arnold (Publishers) Ltd. 1955.

Handbook of Radiology. Edited by Russell H. Morgan, M.D. Pp. 518 + x with illustrations and tables. $10·00. Chicago: Year Book Publishers. 1955.

The Casualty Department. By T. G. Lowden, M.A., B.M., B.Ch., F.R.C.S. Pp. 278 + viii with 170 illustrations. 37s. 6d. Edinburgh: E. & S. Livingstone Ltd. 1955.

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CORRESPONDENCE : BRIEWERUBRIEK

ELI LILLY MEDICAL RESEARCH FELLOWSHIP (SOUTH AFRICA)

To the Editor: I would like to draw the attention of Medical Practitioners registered in South Africa to the fact that applications may now be submitted for the 1955 award of this Fellowship.

The value of the Fellowship is $250 a month for twelve months, plus return travelling expenses to the point of study in the United States of America.

Details of the conditions of the award were published in this *Journal* in the issue of 15 January 1955, at page 67. Information may also be obtained from the undersigned.

The closing date for applications is 15 April 1955.

H. A. Shapiro.
Hon. Chairman,
Selection Committee,
Eli Lilly Medical Research Fellowship (South Africa.)

P.O. Box 7605,
Johannesburg.
2 March 1955.

COLLECTION OF NON-PULMONARY TUBERCULOUS MATERIAL FOR TYPING

To the Editor: The Council for Scientific and Industrial Research is anxious to obtain as many specimens of non-pulmonary tuberculous lesions as is possible, in order to settle the question of bovine infection of man in South Africa.

I should like, therefore, through the medium of your journal, to appeal to any specialist or general practitioner to forward specimens of this material for typing.

The following types of specimen are most needed, the name, age and sex of patient to be indicated:

1. Glandular tuberculosis—neck or elsewhere.
2. C.S.F. from suspected or known tuberculous meningitis.
3. Pleural or ascitic fluid in suspected tuberculosis cases.
4. Pus or bone from any orthopaedic cases.
5. Urine from urinary tuberculosis.
6. Any glands or pus found in laparotomy etc. of tuberculous cases.
7. Any post-mortem non-pulmonary tuberculous material.

Collection of Specimens

(i) *Urine.* Catheter urine is preferred—as large a quantity as possible should be forwarded. In the case of genital tuberculosis, freshly voided urine is satisfactory. These specimens should be sent in screw-capped sterile containers.

(ii) *Fluids.* Pleural fluid, ascitic fluid, synovial fluid. As far as possible the quantity to be sent is 10—15 c.c., to which 1 c.c. of 3% citrate solution has been added to prevent coagulation.

(iii) *Spinal and Cisternal Puncture Fluids.* As large an amount as possible should be forwarded for examination in screw-capped sterile containers.

(iv) *Pus.* 1—3 c.c. should be sent. Small quantities of pus should be sent on sterile wooden swab-sticks. (When being placed in the forwarding tube the swab-stick should be cut with sterile scissors so that the sterile part of the stick is in the tube. A rubber stopper is then inserted in the tube.)

(v) *Tissue, Tonsils and Lymph Nodes.* The material should be sent in a sterile screw-capped container, without fixative.

(vi) *Faeces.* 1—2 c.c. should be sent in a sterile screw-capped container.

The specimens should be sent by quickest route (air freight if necessary) to C.S.I.R. Tuberculosis Research Unit, P.O. Dormerton, Durban.

Your help and co-operation in this matter will be greatly appreciated.

J. J. du Pré le Roux
Secretary for Health

Department of Health
P.O. Box 386
Pretoria
25 February 1955

THE FREEDOM OF THE DOCTOR

To the Editor: Rarely has more sense and saner material appeared in this *Journal* than the report of Dr. J. H. L. Shapiro's presidential address in the issue of 26 February 1955 (**29,** 213).

After having studied the functioning of the medical profession in 4 countries on 3 continents I find that the creeping blight of control moving across the world is silently trying to engulf our profession here too. Almost the whole problem is reflected in what a 'big business' individual said to me a few days ago: 'We employ two doctors and a dentist for our personnel and shall soon employ more; so that we shall not require the medical profession any longer'. This may be somewhat boastful but if we do not take care we shall soon be enslaved by Industry, Big Business and Government.

I, too, feel that the first step towards freedom is to have compulsory membership of the Association and, secondly, compulsory publication in the *Journal* of the policy of candidates for high office. Registration of a vote in our present system is no more than a shot in the dark.

G. P. Fourie

314 S.A. Mutual Buildings,
Cape Town
28 February 1955